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DANGER OF ERROR IN DIAGNOSIS BETWEEN CHRONIC SYPHILITIC FEVER AND TUBERCULOSIS.[1]

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The subject which has been chosen for brief presentation before a medical body composed as this is must be defended by its recital as necessary, or else it would be presumptuous to take your time in the consideration of well-known diseases. A certain number of cases demonstrably of specific nature have come under my observation, condemned as being tubercular, not, as a rule, by physicians of little experience, but by those of well-established reputation, some being teachers and writers of medicine. Let us first pass in review the salient points of such cases, and you will bear in mind that from the nature of the subject more cannot be done.

The first of this group had been sent to a noted health resort for phthisical subjects; but instead of improving had steadily lost ground, so that after three months' trial he came to me reporting a loss of forty pounds in weight, a slight continued fever, a sense of weakness, and a pain in his right side. On examination this pain was found to be dependent upon a perihepatitis which was productive of distinct friction sound, with both inspiration and expiration, and also of palpable friction fremitus. An inquiry disclosed the fact that he had been a victim of syphilis ten years before. No proof could be found of any pulmonary lesion. Under antisyphilitic treatment in two months he had regained his weight, had lost the fever and malaise, and since that

[1] Read before the Society of American Physicians, May, 1898.

time has remained well. No doubt can be entertained that in this case the liver was the site of the specific processes which had been the cause of his hectic and of his ill health. Whether it would have been possible to detect the liver implication at a very early period I cannot determine; but he had seen two of the leading diagnosticians of this country in different cities, neither of whom had made out the nature of his sickness, and by one of them he had been sent to the health resort for tuberculosis of the right lung.

A second case is that of an adult male who came to be examined because of fever, sweating at night, loss of weight, and pain in the right side. He had been ordered by a professor of medicine, who was also a noted examiner of diseases of the chest, to give up his business and remove to a resort for consumptives in consequence of tubercular disease of the right lung. A careful examination failed to reveal any distinct signs of focal pulmonary disease, but on more critical investigation two of the ribs on the right side were found to be sensitive to pressure, and a small sinus was disclosed in the neighborhood of the middle third of the right clavicle. He admitted syphilitic infection in early manhood. Antisyphilitic treatment removed his fever, and within a month he had regained his weight.

The third case relates to a patient who came to me with the history that he had been sent to a prominent health-resort for consumptives because of the nexus of symptoms which have been detailed. After two months' stay there he had been sent home, because of the continuance of his fever and of his steady decline, which, in the opinion of the physician there, thoroughly informed and capable, indicated miliary tuberculosis, for which there was no hopeful outlook. This physician has been frank enough to state that he could not satisfactorily locate the disease. My own examination was negative in so far as making out the location of a process which was productive of the disturbance of his health. This led to a cross-examination of the patient in regard to his antecedents. He admitted that he had had syphilis in the past. Under antisyphilitic measures he made a rapid improvement. Then he removed to another healthy locality, where a little over a year later he suffered from a synovitis of the knee-joint. The surgeons of the place considered this to be tubercular, and advised incision. He returned to me for advice. In addition to the effusion in the joint there was some thickening of the lower end of the femur. Again, under antisyphilitic treatment, within three weeks he had made such improvement as to be practically well, and has continued so since—a period of three years.

A fourth patient was referred to me for probable tubercular implication of the apices of the lungs, who had a rather marked cough in addition to the chain of symptoms outlined. Because no improvement had followed a residence in a mountain resort near his home, and

because of the steady decline in his health, and of the continuance of his fever for five months, notwithstanding the use of quinine and creosote, he had been ordered to a remote mountain region. He came to me to know if I could suggest any plan of treatment by which he could avoid the necessary sacrifice of his business interests should he act upon the advice given. The physical examination of the lungs was negative; such sputum as he could furnish did not contain bacilli of tubercle, neither did the examination of his body disclose any localization of morbid process which could satisfactorily account for the disease. A cross-examination elicited the history of a by-gone syphilitic infection. Within three days from the commencement of antisyphilitic treatment his fever subsided; and he wrote me from his home, after the lapse of three months, that he had regained flesh, strength, and health. His letter closed with the inquiry why his physicians had not resorted earlier to the use of a remedy which had proved so efficacious.

The fifth case was that of a young child who had continued fever, wasting, and a somewhat larger liver than his age warranted. The child had been treated with the idea of malarial infection in vain. The examination of the lungs did not disclose any sufficient evidence of tubercular trouble to warrant a diagnosis of that condition. A careful questioning was made as regards the possibility of tubercular infection through milk or contact, but without proof. The child was somewhat emaciated, and the examination of the abdomen failed to prove any enlargement of the glands. This led to a careful discussion of the case with the child's physician as regards the diagnosis, which, to my mind, lay between syphilis and tuberculosis. The enlarged liver inclined me to the diagnosis of syphilis, but I was assured that there was no possibility that either parent had had that disease, from personal knowledge, and I was dissuaded from making a cross-examination. The result was that a diagnosis of probable tuberculosis was made. Later I was informed that, the child dying, an autopsy had revealed syphilitic infection of the liver, and the parental parent had admitted a by-gone syphilis.

The sixth case is that of a male patient over thirty, now under treatment. He had syphilis ten years ago, and consulted me because of an urticaria which had come from eating stuffed peppers. He had a very pale look, which was dependent on deficiency of hæmoglobin. The examination of the body revealed some enlargement of the liver and spleen. On inquiry, it was learned that he had lost thirty-five pounds in the past two years, had sweats at night from time to time. The result of giving him a thermometer was to disclose the fact that he had a constant temperature which varied from 100° to 101½° for the most part, but on one occasion was 102° and on another 104° F. during eight days of observation. There was more or less sweating at night.

A careful examination of the lungs was made, with negative result. The blood showed neither plasmodia nor hyperleucocytosis. He was placed upon antisyphilitic treatment and five grains of quinine twice daily. Within a week he was much better. Less sweating and only very slight fever were noted. At the end of three weeks he reported that for a week he had not had fever. His weight had increased two pounds, and the examination shows a distinct reduction in the size of the liver and of the spleen. Without administration of iron there had been, moreover, a distinct gain in his appearance, as indicative of improved hæmoglobin hold as the test by hæmoglobinometer.

To these could be added one other, were it not for the fact that an eruption appeared after weeks of fever, for which residence in a hospital, change of climate, and varied medication had been tried in vain. Specific measures produced a cure.

Cases likewise come under observation in which, with a history of past syphilis and a very limited area of lung implication, it can be hoped that the disease may be alone of specific nature. The most instructive case of this kind which I have followed through its course related to a man who came to me after a slight hæmoptysis. The only manifestation of pulmonary trouble was situated in the upper part of the lower lobe of the left lung. Here for a long time slight dulness, a little broncho-vesicular breathing, and a few subcrepitant râles were all that could be made out. No bacilli were found in the scanty expectoration until one year from the attack and after he had been a resident of two health resorts. The disease spread in his case from the small beginning until the whole lower lobe was involved, a cavity formed, and the upper lobe of the same side affected. The question in such a case could be raised as to the nature of the process at its origin, and whether an original specific focus had become infected by tuberculosis. It is, however, manifest that the physical signs enumerated would, in one without a history of syphilis, have been considered sufficient to establish a diagnosis of tuberculosis, considering the existence of hæmoptysis. Moreover, in this case, no impression was made by the employment of antisyphilitic measures.

The consideration of the facts here narrated have convinced me that many physicians are not aware that fever may attend the late manifestations of syphilis, more particularly of visceral syphilis. The neurologist is sufficiently alive to the importance of always considering the possibility of syphilis being an etiological factor in the obscure diseases of the nervous system, and he very generally gives the patient the benefit of a doubt, when uncertain, and attempts a diagnosis by the use of antisyphlitic measures. Moreover, the class of cases to which I have drawn your attention do not go to a syphilologist, but to the general or consulting physician, and this paper has been presented with

the view of directing attention to the necessity of considering syphilis as a possible explaining cause of those obscure phenomena which are usually only considered to import tuberculosis, malaria, or sepsis. Moreover, I do not find that in the presentation of the subject the syphilologist directs attention specific enough to the possibility here presented. Ignorance of the fact that syphilis, in what is termed its tertiary period, may occasion a fever of long duration, malaise, emaciation, perhaps perspiration, also, without of necessity presenting such definite local manifestations, either external or internal, as can be made out on such casual examination as often occurs when a patient seeks advice at the office of a consultant on one or a few occasions, is largely the explanation of the mistakes.

In two of the cases related it was not possible to state the situation of the disease after very careful examination. We must confess that not a few physicians are inclined, with the above assemblage of symptoms, to determine that tuberculosis must exist; and, having reached that decision, enough of that normal variation of physical signs will be made out to lead to the belief that the explanation has been found in certain pulmonary changes. Moreover, it has seemed to me that physicians having a strong belief that tuberculosis existed may have had an auditory illusion or hallucination. It is far wiser and better, to my way of thinking, to hold the mind in such a condition of attention in doubtful cases as shall admit of expression of doubt, than to attempt a positive diagnosis on insufficient data.

THE DIFFUSE INFILTRATING FORM OF SECONDARY MELANO-SARCOMA OF THE LIVER AND ITS ASSOCIATION WITH ASCITES.[1]

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Secondary sarcoma of the liver may occur in the form of circumscribed masses and nodules, as a diffuse infiltration of the whole liver, or in the form of a mixture of both varieties. The reports in the litera-

[1] Read by title at the thirteenth annual meeting of the Association of American Physicians at Washington, May, 1898.

ture show that so far secondary melanosarcoma is the only histological variety of sarcoma that possesses the remarkable faculty of growing diffusely throughout the liver, so that in time the organ becomes almost entirely replaced by tumor tissue. The text-books of pathological anatomy (Ziegler, Birch-Hirschfeld, Kaufman, and others) contain good descriptions of the striking, marmorated appearances of the liver the seat of diffuse, infiltrating secondary melanosarcoma, and pathologists are agreed that secondary sarcoma of the liver, as well as elsewhere, is of metastatic and hæmatogenous origin. In the diffusely infiltrating variety single cells immigrate more or less continuously into the intralobular capillaries of the liver, where they multiply within the vessels and cause a gradual disappearance of the columns of liver-cells by a process of pressure atrophy. These migrating cells may pass through the capillaries of the lungs and probably, also, of the intestines, because in the vast majority of cases it concerns primary melanosarcoma of the eye or its immediate vicinity. As a general rule, secondary tumors develop elsewhere in the body also, especially in the lungs, but the liver seems to furnish the best conditions for the lodgement and further growth of the tumor cells.

As already stated, the majority of the secondary melanosarcomas of the liver are due to metastasis of a primary tumor in or near the eye. A good account of the literature concerning the nature and location of these ocular and orbital melanosarcomas is given by L. P. Hamburgher (*Bulletin of the Johns Hopkins Hospital*, ix., 1898), in a recent report of two cases of secondary melanosarcoma of the liver following sarcoma of the eye; in one case post-mortem showed a nodular metastasis in the liver which weighed 8.3 k. In connection with this report Abel makes some valuable remarks concerning the chemical composition of the pigment produced by these tumors.

The following cases are interesting as typical examples of the diffuse, infiltrating form of secondary melanosarcoma of the liver in their clinical as well as anatomical features; they have an additional and greater value, however, because they illustrate the development of ascites from portal obstruction due to intracapillary proliferation of tumor-tissue in the liver. As far as can be learned from the literature, the development of ascites on this basis has not been noted in connection with secondary sarcoma of the liver. In Musser's case (*Transactions of the Philadelphia Pathological Society*, New Series, vol. i., No. 2) there developed ascites two weeks before death, but this is apparently attributed to a concomitant "dense fibrous hyperplasia" in the liver, which weighed 8.5 k. (17½ pounds), and was the seat of a "round-celled sarcoma with melanosis;" the patient, a man, aged forty-two years, died from the hepatic symptoms two and a half years after the removal of the left eye for melanosarcoma.

CASE I.[1] *Diffuse infiltrating melanosarcoma of the liver, secondary to melanosarcoma of the choroid of the right eye; ascites due to occlusion of the capillaries of the liver and consecutive thrombosis of the intrahepatic branches of the portal vein.*—Man, aged fifty years, manufacturer, of dissolute habits, came under the care of Professor E. L. Holmes, at the Presbyterian Hospital, September 17, 1895, on account of loss of vision in the right eye which dated back about three years. Professor Holmes diagnosticated the condition of the right eye, which was protruding and nodular, to be due to a melanosarcoma of the choroid. Physical examination was otherwise practically negative. On November 15th some ascites had developed, and on the 25th of this month the ascites had become pronounced; there was jaundice and the urine contained bile-pigments. The patient passed into the care of Dr. J. A. Robison, and thanks are due both Professor Holmes and Dr. Robison for their permission to make this extract from the clinical history of the case.

The patient died December 7, 1895, apparently from exhaustion. The post-mortem examination was made one hour after death.

The anatomical diagnosis reads: Melanosarcoma of the right eye and the orbit; atrophy of the right optic nerve; metastatic tumors in the lungs, pleuræ, and left kidney; diffusely infiltrating metastatic melanosarcoma of the liver; chronic congestion of the spleen; thrombosis of the intrahepatic branches of the portal vein; ascites; general bile pigmentation; chronic nephritis; chronic (left) orchitis; general arteriosclerosis.

The body is much emaciated; the skin and visible mucous membranes yellow; the right eye protruding, the eyeball nodular; the orbit is filled with a hard mass; the cornea turbid.

The abdominal cavity contains a large quantity of yellowish fluid; the peritoneal layers are smooth. The pleural and pericardial cavities are empty.

The heart weighs 450 grammes; endocardium normal.

The lungs are œdematous and contain a few small, solid nodules that are grayish and in places almost black on the cut surface. Similar smaller masses are found here and there in the pleuræ. The larynx, trachea, and peribronchial glands are normal.

The spleen is firm, very dark-red in color, contains much blood, and weighs 450 grammes.

The kidneys weigh 350 grammes; the surface is irregular, the cortex thin, the consistence firm. In the left kidney is a small, subcapsular tumor nodule. The left testicle is firm, fibrous.

The liver is very much increased in size, the left lobe being almost as large as the right; it weighs 4100 grammes. The external surface is rather finely nodular, mottled bluish and gray in color; many small, pinhead-sized and a little larger gray tumor masses are seen on the surface. In consistence the liver is very dense and firm, like sole-leather or hard rubber. The cut surface is also irregularly nodular or granular, the predominating or ground color being bluish-black, with small gray districts here and there, so that the general appearance of the surface is best described as granite-like or variegated, like marble.

[1] This case is described in the Transactions of the Chicago Pathological Society, 1897, vol. ii. p. 138.

The large vessels in Glisson's capsule and the branches of the portal vein are filled with rather softly-coagulated blood.

The gastro-intestinal tract is normal, but the mucous membrane is slaty in color. The pancreas is normal.

The aorta and the splenic artery show a rough and irregular intima.

The skull and the brain are normal, the vessels at the base being sclerotic.

The interior of the right eye is partly filled with a firm tumor that seems to spring from the choroid, is quite black on the cut surface, and largely replaces the vitreous humor; at the entrance of the optic nerve the tumor extends for a short distance backward in the substance of the nerve, and at the corneo-scleral junction of the internal aspect of the globe the tumor tissue perforates all the coats of the eye and spreads out into a walnut-sized retrobulbar tumor-mass which is grayish white in color, with only here and there blackish or brownish dots and areas. The right optic nerve is smaller than its fellow, and gray in color.

Blood-serum tubes inoculated from the heart's blood and the ascitic fluid remained sterile.

The microscopic examination of the tumor of the eye and the pulmonary and renal nodules show the structure of a typical melanosarcoma; the pigmentation is most uniform in the eye; in the other places the majority of the cells are not pigmented, but chromatophores occur in all.

The liver shows but remarkably little true liver structure; it consists almost entirely of tumor tissue which is, speaking broadly, very much pigmented, typical, slender, branching cells filled with brownish pigment predominating; often the amount of intracellular pigment is so marked that the nucleus is hard to see. Groups of, and single, non-pigmented cells also occur. Wherever the hepatic structure persist the capillaries are filled with tumor cells often to an excessive degree, producing a marked compression of the adjacent columns of liver-cells, whose gradual disappearance from atrophy can be traced from the early to the final stages, when but narrow, almost imperceptible bands of protoplasm remain, to be succeeded by coalescence of the intracapillary tumor emboli. As a result of this form of growth the tumor has an indistinct alveolated appearance. (The alveolar structure is not nearly so well marked as in the second case, presumably because the infiltration has been more uniform, the embolism and the capillary growth occurring at the same time and with the same degree of rapidity throughout the organ.) Careful scrutiny failed to show any histological appearances that would point to removal of the liver-cells by means of phagocytic action of the cells of the tumor. In the large vessels are often found single cells, as well as masses of tumor cells. Remnants of, as well as intact, biliary ducts are found, principally in a somewhat thickened Glisson's capsule which is the seat of a small degree of round-cell infiltration. Otherwise there has been no connective-tissue increase in the liver. In some of the larger vessels are hyaline thrombic masses, the exact nature of which is doubtful, because the pieces examined were hardened in Müller's fluid. In some places the tissue in what must be parts of Glisson's capsule presents an appearance as though it were œdematous.

CASE II. *Melanosarcoma of the eye, following trauma; extirpation and*

recurrence; secondary infiltrating sarcoma of the liver, with ascites.—Herman K., native of Germany, a resident of Cook County, Illinois, for eighteen years, by occupation a farmer; entered Dr. Herrick's ward in the Cook County Hospital, September 22, 1897.

Of his family history he knew little. There was no hereditary taint, so far as he knew. He himself had been temperate as regards the use of liquors, had escaped venereal infection, and had been singularly free from preceding serious illness.

He was more surprised, therefore, to find that during the past year, and particularly during the last six months, he had been losing in flesh and strength. His chief complaint was of this weakness and of a feeling of fulness and hardness in the epigastrium that had annoyed him for about four months. For one week there had been considerable distention of the abdomen. He complained of no pain whatever, had a good appetite, never vomited; bowels were regular.

Inquiry as to the circumstances leading to the loss of his right eye elicited the information that three years before he had been kicked over the right eye by a horse, the eye, or the parts in the immediate vicinity, becoming greatly swollen. This swelling soon subsided, and the eye gave no further trouble until two years after, when it became swollen, "red, like a beefsteak," and the sight failed. The eye was removed at the Illinois Charitable Eye and Ear Infirmary one year prior to his admission to the County Hospital.

Status præsens. There is marked emaciation, the skin is dry and scurfy, of a muddy look; there is no icterus. The movements are evidently painless, though he is plainly very weak. The breath is fetid, the teeth decaying.

The left eye is normal, the right eye lacks the globe. Beneath the drooping lid a small, hard nodule can be felt, and the muco-purulent discharge comes from a reddish mass of granulations, and is often blood-tinged.

The lungs are clear. Over the lower right chest the dulness, continuous with that of the liver, rises in front and behind one inch higher than normal. The heart's action is rapid and snappy; its apex impulse is in the fourth interspace close to the nipple, as though displaced by the enlarged liver. The arteries are tortuous and have slightly thickened walls.

The abdomen is distended, and the signs of free fluid can be made out. The liver is plainly palpable, in the mammillary line its border being a hand's-breadth below the costal arch. Dulness in front is to the fourth rib. The surface of the liver feels firm and coarsely granular; it is not tender. In the epigastrium there is a distinct bulging forward. The edge is somewhat irregular or scalloped and is very hard. No nodules are anywhere distinctly made out. The liver descends freely with inspiration. The firm hard mass of the liver, the patient tells us, has been noticed by him for about four months.

No change can be made out in the other abdominal organs. The stomach is not enlarged; its contents were not examined. The rectum and genitalia are negative. The umbilicus is prominent. The inguinal glands are, perhaps, slightly enlarged, as are the axillary. There is œdema of the ankles and legs and slight œdema of the abdominal wall.

Scattered over the surface of the chest and abdomen are several hard, painless, freely movable nodules apparently in the subcutaneous tissue.

The largest, the size of a large lima bean, is just inside the vertebral border of the left scapula. The color of some of the nodules, as seen through the skin, is slightly black. One of these nodules in the abdominal wall was excised for purposes of diagnosis. This showed small round- and spindle-celled structure, with considerable fibrillated stroma; in occasional areas the cells contained small granules of brownish-yellow pigment, which also occurred between the cells. Diagnosis: Melanosarcoma.

The urine contained neither sugar, albumin, nor casts. When fresh it was clear, of a reddish-yellow color, and acid. On standing for several hours it became of a darker hue, and still remained acid.

With strong sulphuric acid and with nitric acid the color changed to a dark brown. On the addition of a small amount of a strong solution of ferric chloride a grayish precipitate was produced, which dissolved in an excess of the reagent. With bromine water a yellowish precipitate that gradually darkened was formed. These tests were regarded as evidence of the presence of melanogen in the urine, a common finding in cases of melanotic growth. In every instance the test was controlled by similar tests with other urines.

The diagnosis was made, therefore, of melanotic sarcoma of the eye, with metastases in the liver and subcutaneous tissue, and with recurrence of the growth at the original site. This diagnosis was further confirmed when an examination of the records at the Eye and Ear Infirmary showed that the eye had been removed for melanotic sarcoma. Careful examination was made for evidences of involvement by metastases of the lung, pleura, and pericardium, but neither by symptom nor by physical sign of friction, dulness, or altered respiratory or circulatory tone, was there any revelation of the existence of the nodules in these organs. The dulness from the small amount of fluid in the right pleura was believed to be due to the enlarged liver.

The question naturally arose as to the condition of the liver and the cause of the ascites, whether we had to deal with a pure sarcomatosis of this organ or with sarcoma in a cirrhotic liver. Notwithstanding the fact that the statement was made by some authors—*e. g.*, Leube—that in sarcoma of the liver ascites does not occur, the fluid in the abdomen was regarded as probably due to the growth in the liver, and this, from the absence of distinct nodules in a liver of these enormous proportions, was looked upon as probably the diffuse infiltrating variety. The solution of the clinical problem was rendered much easier by the recollection of the specimen of diffuse infiltrating sarcoma of the liver associated with ascites, shown by one of us at the Chicago Pathological Society a short time before (Hektoen: *Transactions Chicago Pathological Society*, 1897, vol. ii. p. 138.). That the fluid in the abdominal cavity was a transudate seemed probable from the fact that on October 9th a pint of clear serous fluid was withdrawn by puncture, and its specific gravity was but 1007. In the fresh and stained specimens from this fluid no pigment-containing cells were found. Nor were any cells resembling sarcoma cells or large cells with the atypical mitoses described by Rieder, Dock, and Warthin found. The peritoneum was presumably free from tumors and the fluid due to portal obstruction. The patient died rather suddenly a few days later.

Anatomical Diagnosis: Absence of right eye; pigmented tumor in right orbit growing into optic nerve; pigmented and pigment-free

tumors in the skin, the muscles, the lymph nodes, the pleura, the lungs, and the myocardium; diffuse tumor infiltration of the liver; ascites; right hydrothorax; left adhesive pleuritis, with slaty induration of left apex; œdema of the legs; ecchymoses in the floor of the fourth ventricle; sclerosis of the coronary arteries and of the aorta; chronic nephritis.

Abstract of Post-mortem Protocol: The body is wasted. The right eye is absent, and the orbit is filled with a nodular mass which is blackish on the cut surface. In the skin of the anterior surface of the chest are three or four movable, split-pea sized, slightly pigmented nodules; in the muscles to the left of the dorsal part of the spine is an oval tumor, 2 by 4 cm., with areas of blue pigmentation.

There are small pigmented masses in the anterior mediastinum; considerable clear fluid in the right pleural cavity; the left is obliterated by adhesions. In the pulmonary and diaphragmatic pleura are innumerable pedunculated and sessile tumor masses of varying size; there are tumors in the lung substance and in the peribronchial glands.

In the epicardium are many tumor nodules, the largest being over the anterior wall of the right ventricle near the coronary sulcus; the nodules are grayish or whitish in color. Attached to a papillary muscle in the left ventricle is a minute, pedunculated black mass. The coronary arteries and the aorta are somewhat sclerotic. The heart weighs 300 grammes and the endocardium is smooth.

The abdominal cavity contains several pints of clear, yellowish fluid; the peritoneum is smooth and shining. There are no thrombi in the portal vein or its large branches. The mucous membrane of the intestines is normal, that of the stomach rather slaty in color. In the omentum and mesentery are many small nodules.

The liver weighs 5000 grammes; it is the seat of a uniform enlargement that seems to involve the right lobe more than the left; the surface is mottled-gray and bluish-gray or black, and is quite smooth for the most part; but over the left lobe and along the lower margin of the right it is rough and even nodular, due to grayish or bluish projections; the consistence is very firm. On the cut surface the left half of the liver has a more bluish color, the right half a more yellowish gray, the line of junction being in some parts very sharp and recognizable on the external surface, but mottled areas of variegated appearance like marble also occur, as well as distinct nodules of varying sizes. Any traces of normal liver substance are not to be seen. The gall-bladder contains a small amount of bile. The pancreas, the spleen, the genito-urinary organs, and the adrenals are free from tumors; the surface of the kidneys is finely granular, the consistence increased. In the floor of the fourth ventricle are small hemorrhages; the tumor in the orbit extends in the optic nerve to the commissure, filling the nerve with a bluish-black mass.

The Microscopical Examination: Pieces from all the tumor masses were examined after the usual technical preparations. The pigmented as well as pigment-free masses outside the liver may be dismissed with the general statement that in the main they are made up of rather closely-packed, small, spindle and round cells, that run in interlacing bands of varying thickness which, cut across, give the tissue an alveolar appearance; here and there occur characteristic chromatophorous cells in small groups or scattered about singly.

A number of pieces were studied from different parts of the liver, in order to obtain a clear notion of the relation of the tumor-cells to the structural elements of the organ. It may be said at once that almost the whole liver has become substituted with tumor whose further expansive growth has been limited and so directed by the original framework of the liver that the resulting mass, at the time of death weighing 5000 grammes, still preserves the outline of the liver. It concerns, therefore, a diffuse infiltrating sarcoma of the liver.

Here and there in the uniform infiltration occur, as seen with the naked eye, distinct nodules. In these rather vascular areas the structure is indistinctly alveolar, the spaces being closely packed with rather small, mostly pigment-free, sarcomatous cells. A few small multinucleated cells are seen. The pigmented cells are mostly long drawn out, branching, even star-shaped, and also round; the pigment is yellowish or deep brown, mostly granular, sometimes scaly, at times covering all parts of the cell densely; at other times it is gathered along the borders of the cytoplasm or at the ends of the processes. When the processes are cut off, free pigment heaps are simulated. The pigment cells seem to prefer the outskirts of the spaces and to lie in the more distinct fibrous bands. Their shape and the arrangement of pigment granules are best seen in unstained preparations.

In the flat infiltration the alveolar arrangement of the cells is very prominent. The spaces are of various size, round, oval, more rarely oblong; the walls of the spaces are sometimes very indistinct, sometimes made up of a distinct layer of fibrous tissue, with narrow, elongated nuclei; again, of endothelioid cells, and finally remnants of bands of liver cells may be met with at the limits of the alveoli. The cells in the alveoli are mostly pigment-free, oval, round, or polygonal, with rather small bodies and deeply-stained nuclei; a few chromatophorous cells are frequently observed, and in some districts they predominate. Now there are extensive areas corresponding to this general description without any further trace of liver structure, and but few bloodvessels; occasionally, bands of hyaline connective tissue may be seen, with vessels and, perhaps, two or three rows of low columnar cells without any arrangement to form any distinct lumen; distinct, but empty, bile-ducts also occur.

The early stages of this replacement process are clearly made out on studying parts where some distinct liver tissue still persists.

In the first place, it is noticeable that of the capillaries encountered, all, or almost all, contain tumor cells among the blood-corpuscles or free in the empty lumen. The majority of these cells are chromatophorous, and the long, branched, star-shaped form of this cell is seen most typically among the blood cells. The majority of the persistent capillaries and vessels are unduly congested. From this beginning there can readily be traced a progressive intracapillary growth of melanosarcoma which crowds out the blood, distends the capillaries to the utmost, and produces a pressure-atrophy of the liver cells. In the earlier stages the rows of liver cells are still distinct, but the cell bodies soon lose their outlines and become amalgamated into protoplasmic bands that stain deeply with eosin and are thickly set with nuclei; the bands become thinner, the nuclei smaller and more closely crowded together until the resulting alveoli of tumor cells come into contact with each other. From the histological appearances it seems that a purely

mechanical pressure-atrophy of the liver structure precedes its often complete replacement with tumor tissue.

While the early capillary tumor-cell emboli seem to be principally chromatophorous, the more advanced intracapillary proliferations may in some cases produce pigment-free cells in predominating numbers; hence this may account for some of the extensive non-pigmented areas which, as seen on the cut surface of the liver, are most sharply demarcated from the pigmented districts; as already remarked, the process is everywhere so far advanced that nothing can be said as to just in what part of the lobule the free cells become first arrested, nor can any exact details be made out with respect to the hepatic veins or smaller bile ducts.

In a number of pigment-free or but slightly pigmented cells are karyomitotic figures.

It is, finally, noticeable that any proliferation and induration of the fibrous tissue has not occurred to the slightest degree.

CASE III. *Melanosarcoma of the right eyelid; extirpation; secondary sarcoma of the liver, presumably infiltrating, of the pleura and of the peritoneum, with ascites; no autopsy.*—Joseph P., aged fifty-four years, German, laborer. Admitted to Dr. Herrick's ward in the Cook County Hospital. Family and personal history negative.

In May, 1897, a dark-colored, easily-bleeding nodule of two years' standing was removed from the right upper eyelid—together with one-half of the lid—by Dr. D. A. K. Steele. February 28, 1898, patient felt so weak that he gave up work, which was loading coke. He had lost in weight, was short of breath, and was distressed by the increasing size of the abdomen.

Examination March 22, 1898, in Cook County Hospital, showed emaciation and weakness. One-half of right upper eyelid was left and showed nothing abnormal. Both eyes were the seat of a conjunctivitis, with a muco-purulent discharge. No growth could be demonstrated in the eye. Vision was good. Several dark-colored nodules, intimately connected with the skin, were scattered over the body, the smallest about the size of a pinhead, the largest, situated in the scalp over the right parietal region, as large as a hickory-nut. The lungs and heart were negative. There were signs of fluid in the right pleural cavity; by aspiration clear, light-yellowish, serous fluid. Cells containing fat and brownish pigment found in the sediment. There was free fluid in the abdominal cavity; 6500 c.c. were withdrawn. It was clear, yellowish, specific gravity 1018. Cells of varying size, often with two or three nuclei, and many containing pigment, were found in this fluid.

After the withdrawal of the fluid the liver could be palpated, its edge extending nearly to the umbilicus. The enlargement was chiefly in the right lobe. The surface and edge were hard, not nodular. In the right half of the epigastrium the enlargement caused a visible bulging. The spleen was not palpable. No masses or nodules could be felt in the abdomen. The lower extremities were not œdematous.

The red blood-cells were of normal size, shape, and color. They numbered 4,500,000 per cubic mm.; white cells 9680, with slight polymorphonuclear increase. No abnormal pigment was seen in the blood in fresh or stained specimens. Hæmoglobin, 80 per cent. (Fleischl).

The urine was scanty—fifteen ounces for the twenty-four hours. It

was acid, specific gravity 1030. No albumin, no sugar, no casts. Tests for melanogen with sulphuric acid, bromine water, and ferric chloride, all positive, and controlled by tests with known normal urine.

The diagnosis seemed clear of a pigmented growth involving the liver, skin, pleura, and peritoneum, and presumably secondary to the primary growth removed in May, 1897, from the eyelid, which growth had existed for two years. To confirm the diagnosis and to determine more accurately the nature of the tumor, one of the nodules from the chest was removed and examined microscopically. This showed a fibrillated stroma with an alveolar arrangement, packed with small round and spindle cells. Reddish-brown pigment granules and masses were seen here and there, having a tendency to be arranged in the fibrillary tissue and parallel with the fibres, but being found also in and among the round and spindle cells. Diagnosticated as a melanotic sarcoma. It was further learned from Dr. Steele's clinic that the diagnosis of the tumor of the lid at the time of the operation had been melanotic sarcoma. Unfortunately the patient refused to stay in the hospital and left at the end of a few days.

Remarks. In general, the structure of the tumors agrees closely with that laid down by Hugo Ribbert (*Ueber das Melanosarkom, Ziegler's Beiträge*, 1897, xxi.) as peculiar for the melanosarcoma, or melanoma, as he would prefer to call the tumor in question, of the eye. The essential structure of the various growths is identical. The presence of the characteristic pigment cells in the majority of the areas, although in greatly varying numbers and degree of pigmentation, demonstrates clearly enough that it concerns metastatic tumors secondary to melanosarcoma in these cases of the right eye. Of the secondary growths, especial attention is called to the diffuse infiltrations in the liver. The first two cases, and probably the third, are typical instances of the diffuse infiltrating variety of secondary sarcoma of the liver, which nearly always concerns melanosarcoma. The histological examination points to a remarkably extensive and uniformly diffuse capillary embolism of tumor cells. In one case these cells passed almost unhindered through the pulmonary capillary network, as only a few tumor nodules were present in the lungs. In the second case cells were arrested in the lungs, the pleura, the myocardium, the pericardium, the skin and subcutaneous tissue, and an implantation nodule developed on the endocardium of the left ventricle, but in both cases the capillaries of the liver afforded the best conditions for arrest and further growth and proliferation of the cells in the blood. Apparently the cells reached the liver through the hepatic artery rather than through the portal vein, because there were no metastases in the capillaries drained by this vein, but in view of the uncertain and accidental manner in which secondary tumors may arise, only conjectures in regard to this point are permissible. In the liver, however, the favorable conditions for cell-growth resulted in the development of a diffuse intracapillary growth, which replaced the

liver tissue proper while it largely retained the normal external conformation of the organ. In both cases this process is everywhere so far advanced that it cannot be said in what part of the lobule the cells were first arrested. As indicated, the capillary tumor cell emboli seem at first oftenest chromatophorous; as these cells proliferate large numbers of pigment-free cells are produced, and there result in this manner large districts, and in one case more distinct nodules also of non-pigmented tumor growth. It is a well-known fact that the cells of a metastatic melanosarcoma tend to lose their original physiological property of producing pigment. Ribbert looks upon the round, non-pigmented cells in such foci as incompletely developed. An exact explanation of the origin of extensive pigmented and non-pigmented areas with well-marked, definite margins cannot be advanced; it is easy to suggest that this feature, which was well marked in the second case, may depend upon accidental distribution of pigmented and pigment-free cell emboli.

There is absolutely nothing in the histological appearances, which are quite plain, that would warrant the inference, advanced by some, that the melanotic cells exercise a proliferative or "infectious" stimulus upon the cells with which they come in contact, leading to an active participation of pre-existing elements in the growth of the secondary tumor tissue; on the contrary, it is quite plain that the metastatic process that has led to replacement of the liver with tumor tissue is one of continuous intracapillary tumor growth, resulting in, so far as can be specified from the microscopic appearances, a mechanical pressure atrophy of the intervening liver substance associated with compression and occlusion of the structures in Glisson's capsule. As the capillaries and small vessels become packed with tumor cells the portal circulation is hindered, ascites develops, and, in one case, thrombosis of the intrahepatic branches of the portal vein was induced. The development of ascites upon this basis—*i. e.*, occlusion of the hepatic capillaries by sarcomatous cells, to which attention was drawn in the report of the first case (*Transactions of the Chicago Pathological Society*, 1897, vol. ii.)—is exceedingly interesting, and does not seem to have been previously mentioned in the descriptions of this remarkable form of secondary sarcoma of the liver.

These cases deserve notice from a clinical as well as anatomical point of view.

In Case II. there was a clear history of the local trauma two years before the removal of the eye. This is so common in cases of melanosarcoma as to attract attention in all series of cases. Lawford and Collins[1] found local injury several months or even three years before the

[1] Lawford and Collins: Sarcoma of the Uveal Tract, with Notes of 103 Cases. Royal London Ophthalmic Hospital Reports, 1891, p. 104.

removal of the eye for melanosarcoma in 6.79 per cent. of cases. P. Wagner,[1] in nineteen cases of melanosarcoma, reports a history of trauma in five.

The frequency with which the liver is secondarily affected is commented upon by all writers. In our three cases the involvement of the liver was at the time of observation the predominating feature.

In Case III. the primary tumor was apparently in the eyelid, an unusual locality.

In the matter of diagnosis, we would emphasize the importance of careful inquiry as to the nature of existing eye troubles and of causes for extirpation of eyes seen to be missing. Further, painstaking search for cutaneous and subcutaneous nodules should be made. Many of these are extremely minute and to be detected only by diligent examination, their existence not being known to their possessor.

The extirpation of one of these nodules for microscopic examination is a simple matter, but of great aid in diagnosis.

While other causes for melanuria than melanotic neoplasm are not unknown—*e. g.*, malaria—the presence of melanuria or melanogen in the urine adds strong confirmatory evidence of the existence of a melanotic growth, in the liver, for example, if that be enlarged.

Melanogen is a colorless chromogen that first becomes black by oxidation, and is then called melanin (Litten). A single negative examination of the urine should not be regarded as excluding a pigmented tumor, for in some cases neither melanin nor its chromogen has been present, and in others the melanuria has been periodical. We would emphasize the value of control-tests with normal urine as greatly limiting the possibility of error. The question of the nature of these pigments in the urine is fully discussed by Litten. (Litten : " Ueber einen Fall von Melanosarkom der Lieber." *Deutsch. med. Woch.*, January 17, 1889.)

As to the configuration, size, and consistency of the liver, it may be said that in the diffuse infiltrating form of sarcoma the liver seems to preserve nearly the normal shape, though with its measurements greatly increased. It is hard, and if the abdominal wall be thin enough may be felt to be slightly roughened or coarsely granular. In the mixed form the surface may be uneven from nodules, and these may be hard or soft, fluctuating, and containing fluid.

The occurrence of ascites should not, as some authors state,[2] argue against sarcoma of the liver. Its clinical association without peritoneal origin is demonstrated by Cases I. and II., and the cause seems not obscure when we consider the pathological condition present, the plugging of the hepatic capillaries by sarcoma cells.

[1] P Wagner: 19 Fälle von Melanosarkom. Münch. med. Woch., August 16, 1897.

[2] Leube lays stress on the absence of ascites, " which, in sarcoma of the liver, is apparently invariably lacking." Tyson also makes a somewhat similar statement.

In our second case the small omental nodules did not seem a sufficient explanation of the fluid, which further possessed the characteristics of a transudate; specific gravity 1007. In our first case no cause for the ascites save the hepatic growth was discovered. In Litten's case, which was nodular, to be sure, there is no mention of involvement of the peritoneum, yet there was a moderate amount of ascitic fluid in the lower part of the abdomen. In our third case we believe, from the character of the fluid, that the peritoneum itself was the site of multiple metastases. That in sarcoma of the liver other causes for ascites than plugging of the vessels by sarcomatous masses may exist is, of course, true. A portal thrombosis, a sarcomatosis of the peritoneum, an accompanying nephritis or hepatic cirrhosis—as in Musser's case—may give rise to ascites. But the fact should not be overlooked that ascites may occur, and often does occur, in uncomplicated sarcoma of the liver, at least in the secondary infiltrating melanotic form.

We would add a word as to the value of examination of the fluid aspirated from the serous cavities in cases of suspected involvement of the lining membranes in melanosarcomatosis. In Case II. no pigmented cells were found, nor any cells of unusual size or shape or with nuclei showing atypical mitosis. The fluid was, further, of low specific gravity —1007—and resembled therefore in every respect a transudate. In Case III., however, where we believe the peritoneum was involved in the new growth, not only was the fluid of higher specific gravity, 1018, as in the exudates, but the cells were much more numerous, and many of them were large and in all stages of direct division and some revealed karyokinetic figures. Many cells contained reddish-brown pigment granules, as well as fat-drops.

The absence of pain and of gastro-intestinal disturbances and the slight alteration in the blood-findings seemed in Cases II. and III. rather remarkable, when we consider the advanced stage of the disease; for usually great disturbances in appetite and digestive power are met with in advanced malignant growth, and marked deterioration of the blood is commonly found in these conditions.

SUBARACHNOID SEROUS EXUDATION PRODUCTIVE OF PRESSURE SYMPTOMS AFTER HEAD INJURIES.

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The practitioner not infrequently meets with cases of head injury in which moderate febrile movement and prolongation of unconsciousness, greater or less in intensity, tend to eliminate simple concussion, while

symptoms of irritation, combined with those of depression, unaccompanied by focal signs, point to more or less diffuse cortical lesion. If the discussion were limited to this class of cases it would be perhaps of no vital moment whether the diagnosis of contusion, bruising, or laceration were adopted, provided only the general condition is recognized, and no active steps are taken in treatment. When, however, to this symptom-complex is added a localized paralysis, whether affecting an arm, a leg, or the entire half of the body, and when, in addition, the depth of unconsciousness, for the first day or two at least, is sufficient to cause anxiety, the question of operative interference at once suggests itself. In such cases the importance of most definite ideas regarding the pathology becomes obvious. The autopsy, unfortunately, furnishes comparatively little clue, so that we are forced to rely somewhat upon theoretical considerations, supported as far as may be by actual observation on the operating-table.

It not infrequently happens in these cases that trephining over the area indicated by the paralysis shows negative result beyond revealing a tensely bulging dura, incision of which is followed by free flow, or rather gush, of clear fluid, the brain beneath presenting no laceration or other abnormality beyond, perhaps, œdema.

A few words will be here in place regarding the seat and purpose of the cerebro-spinal fluid. For the purposes of this discussion we may designate the arachnoid as a delicate membrane situated inside the dura and outside the visceral pia. This membrane closely approximates and is hardly separable from the visceral pia over the convolutions, but bridges over the gyri, and at the base of the brain is widely separated from it, leaving a very considerable space, the same conditions obtaining between the cerebellum and the bulb.

Under this membrane in the interval commonly designated the subarachnoid space the greater part of the cortical cerebro-spinal fluid is collected. Increase of cerebro-spinal fluid not only plays a compensatory part in case of lessened blood-pressure and cerebral atrophy, but results also from increased blood-pressure, as in meningitis, and again serves to relieve, according to Tilleau, the intense engorgement following forced expiratory efforts, as, for example, that produced by prolonged coughing. The transudation of this fluid under normal circumstances, as during tranquil respiration and ordinary cardiac systole, is, of course, accommodated for by nature and produces no symptoms. Under abnormal circumstances, however, the bulk of fluid is capable of rapid alteration, as a preservative effort on the part of nature, whether to relieve vascular tension on the one hand or to fill a vacuum on the other.

The transudation of cerebro-spinal fluid is certainly a very rapid process, and no less rapid is the exudation of serum in the cuticle after bruising, whether the fluid shows itself by infiltration of the tissues or

by the localized cavity formation known as a vesicle. There is no reason to suppose that the brain is less liable than other tissues to show congestion and œdema after local bruising, or that an exudatory effort on the part of the bloodvessels would be more tardy than elsewhere in a region in which relief of congestion is of vital moment. Whether the accumulation of fluid following a bruise of the brain has simply the characteristics of cerebro-spinal fluid, or whether it partakes more of the nature of serous exudation, is an easy matter to verify by chemical analysis. The opportunity has not presented itself to the writer since commencing this communication, but we may safely assume the process to be exudatory. There is in either event no theoretical reason why rapid local increase of subarachnoid fluid, with or without accompanying œdema of the brain substance, should not follow local bruising and swelling of the cortex, or its general increase follow diffuse injury. We have, in point of fact, something more than theory to rely upon, such accession of fluid being not infrequently demonstrated by operation.

That a more or less diffuse œdema, whether involving the subarachnoid cavity or the brain itself, or both, and whether accompanied or not by marked laceration, may play a part in prolonging the general symptoms of concussion, seems a not improbable supposition; it is not, however, of this class of cases that I would speak at length, but rather of those cases in which local paralysis of more or less temporary nature accompanies such general symptoms, the pathology being here, I apprehend, a local accumulation of fluid under the arachnoid resulting from rapid exudation at the point of most marked bruising, whether the result of direct violence or of *contrecoup*.

Park has drawn attention to the close adhesion of the arachnoid to the pia at the vertex, excepting where the former membrane bridges the gyri, and as a caution in cerebral irrigation he mentions a case of Macewen, in which about two ounces of fluid were injected apparently into the subarachnoid space and retained. In this case a pressure paralysis ensued and lasted four days. This striking though inadvertent experiment absolutely demonstrated the fact that fluid may remain practically imprisoned under the arachnoid, the condition exactly approximating that of a blister of the cuticle.

Local cerebral œdema has been already suggested (Edes) as sufficient cause for temporary paralysis in medical cases, and Bullard especially has called attention to the fact that the pressure of such œdema may extend, in hemorrhage, the paralysis beyond the region implicated in the original lesion, but little attention has been paid to such pathological conditions as I have indicated, resulting from localized trauma. Trauma has been mentioned, *e. g.*, by Quincke[1] among other causes for serous

[1] Quincke: Volkmann's Sammlung klin. Vortrage, N. F., 1893, No. 67; also Deutsche Zeitsch. f. Newenheilkunde, November 12, 1896, 9 Band, 3 and 4 Heft.

meningitis. The few cases cited by the author, however, in which trauma played a part were of a rather different type from these cases to which I would call attention, the symptoms of pressure being more marked, the optic nerve even becoming atrophied in one case, and blindness following. Lumbar puncture in this case showed marked increase of pressure and a very considerable escape of serum with specific gravity 1008. In another case of chronic hydrocephalus, with acute exacerbation and death at the age of thirteen years, the influence of a fall at the age of five years was very doubtful.

The extremely suggestive work of this author deals in general with internal rather than external increase of fluid. Still the analogy is striking between the symptoms of Quincke's serous meningitis and those of the cases under discussion. Slight febrile movement at the onset of acute cases, headache, restlessness, and vomiting (not constant), dulled sensorium, with or without delirium, sluggish pupils, and occasional local paresis, characterize both. The variation in intensity of symptoms is similar. The frequency of optic neuritis, however, followed by atrophy, in Quincke's cases, together with the retraction of the head, affords a decided contrast to these cases under discussion, while, on the other hand, the complete paralysis leading to suspicion of hemorrhage does not appear to be noted in the cases of typical serous meningitis. In fact, lack of definite local symptoms constitutes one of the diagnostic features of the latter disease as distinguished, for example, from tumor.

In the following fairly typical example of the class of cases to which I will call attention the symptoms were so far suggestive of hemorrhage as to lead to serious question of operation:

CASE I.—A boy of six years, with a negative history, excepting that he is said to have had meningitis and hydrocephalus at eighteen months, and that he is said not to be particularly forward in school training, was struck by a bicycle at noon. Was dazed but not unconscious. That evening he had fever and vomited. He slept until midnight naturally, was then restless until daylight, and at 9 A.M. vomited again. At 1 o'clock the next day, when seen by Dr. Percy, the attending practitioner, he was drowsy, and during the afternoon became unconscious, paralysis developing on the right side, including the arm, leg, and face. The right pupil was larger than the left, but reacted to light. The knee-jerks were present, and the plantar reflex was more active on the left. Defecation and micturition were involuntary. There was a contusion behind the left ear, pressure upon which caused him to cry out.

At 9 P.M., when seen by Dr. Scudder and myself with Dr. Percy, the temperature was 102°, the pulse 96. He could be roused more easily than at six o'clock, and at times spoke coherently when questioned. The facial paralysis was less marked, and the right arm and leg were moved somewhat.

The case up to this time had appeared very suggestive of middle meningeal hemorrhage, and if his condition had steadily grown worse instead of slightly improving, at this time operation would have been

strongly urged. In view of the improvement, and in consideration of the age of the patient, it was decided not to operate at once, but to have him removed to the hospital, prepared for operation, and carefully watched.

On being brought to the hospital at 11.30 P.M., he was still drowsy, but could be roused a little and asked for water. The pupils had become equal and reacted. The pressure-symptoms had already lessened, the right arm and leg being moved, though not so freely as the left. There was still slight facial paralysis on the right. The knee-jerks were now absent. Behind and a little above the left ear was an area of great tenderness, with œdema of the scalp. No depression was found. The patient was put to bed, with heaters, and a special nurse appointed to watch for increasing coma, paralysis, or spasms.

During the night the patient was restless and turned constantly. In the morning the paralysis had lessened still more. He was much brighter, and took liquids. A slight ecchymosis appeared behind the left ear. Some headache was present. Dejections were involuntary. On the second day of his stay in the hospital—that is, the fourth day after the accident—he was restless and fretful during the night, crying out at times with headache. He was given ten grains of bromide of potassium and an ice-bag applied. Was still somewhat drowsy, but inclined to talk at times. The pressure symptoms had disappeared. On the fourth day of his stay at the hospital the child was apparently well, and eating and sleeping well. He was kept under observation ten days longer, during which time an acute inflammatory throat trouble appeared, evidently a coincidence, and he was discharged apparently well two weeks after admission.

Dr. Percy tells me that about three weeks later diffuse swelling of the left side of the face appeared and persisted for a week. The swelling included the lids and extended to the neck and behind the ear. There was no discoloration except in a slight degree under the eye. Whether this symptom bore any relation to the accident I shall not attempt to decide.

Another case of temporary paralysis, occurring in a child, was seen by me with Dr. Boland, of South Boston, who has kindly furnished the following report:

CASE II.—L. D., aged three and one-half years, was thrown to the floor from a child's doorway swing. Her mother was swinging her carefully, when one end of the rope slipped from the suspending hook, and the child was thrown to the floor, striking on her head. The mother was alarmed because the child only moaned and did not cry. I saw the child within an hour and found her dazed. I gave a good prognosis. However, the mother soon noticed that the child could not hold her head up, did not take notice, though she could be roused, and spoke "Mamma" appropriately, also that she vomited. The next day it was noticed that her left arm was useless, and the left occipital region was tender, or at least she flinched when it was pressed, though no ecchymosis or swelling could be seen. The following day Dr. Walton saw her with me. She was so constipated that I had to give her high rectal enemas to secure an operation.

About three days after the injury it was deemed best to send her to

the Boston City Hospital, with a view to operating if paralysis persisted. Over-crowding caused a delay of twelve hours, and then it was noticed that she began to use her left hand. From this on her convalescence was rapid, and she soon showed no remains of the accident. She is now a bright, pretty child of five and a half years, and is as bright in school as any of her age.

This patient also narrowly escaped surgical interference, my hesitation in advising operation arising rather from general considerations than from a definite idea of the pathological process.

I am now inclined to believe that the lesion in each of these children was localized subarachnoid effusion, resulting from a bruise, and followed by rapid absorption of the fluid.

For absolute demonstration that such accumulation of fluid can take place we have to depend rather on the operating than the autopsy-table, and the following case, reported in full by Dr. Brooks and myself in the *Boston Medical and Surgical Journal*, presents a sufficiently obvious illustration:

Case III.—A young woman, thrown from her horse, struck her head violently against a rail, and was carried home unconscious. Four hours later was still unconscious, breathing quietly, with a pulse of 100. The pupils were alike, somewhat dilated, and reacted sluggishly. There was partial paralysis of the left side of the face, and complete paralysis of the left arm and leg. The patient had vomited after the accident. There was a hæmatoma in the right mastoid region. The patient was taken to the hospital.

Restlessness supervened, and consciousness partially returned. Respirations were shallow; there was incontinence of urine. The left arm and leg became rigid.

On the second day the rigidity of the left arm and leg became less marked, and limited voluntary movements appeared. No marked improvement having taken place, however, at the end of thirty-six hours, it was decided to trephine.

Operation. A small trephine button was removed about two and a half inches above the external auditory meatus. The dura, which was in close proximity to the skull, was slightly nicked, and a quantity of clear serum escaped from the opening. The dura was tense and bulging, and on its incision about half an ounce of clear serum spurted out. The brain appeared somewhat œdematous and prominent, but otherwise normal.

On the third day the left arm and leg moved somewhat and were less rigid. The patella reflex was still absent. On the fourth day the movements of the left arm and leg exhibited further improvement, and the patella reflexes had returned. There was less difference between the two sides of the face.

On the fifth day the left side was used much better, and there was little, if any, facial paralysis. From this time on the general condition varied from moderate delirium to somnolence, a fair amount of nourishment being taken by the mouth. The patient gradually sank, and on the fifteenth day the temperature, pulse, and respirations rose rapidly and she died on the sixteenth day. The autopsy revealed no gross lesion

sufficient to explain the fatal termination. Two hemorrhages about the size of beans were found in the left frontal lobe, and various minute hemorrhages were scattered over the brain.

The mechanism of death in this case is somewhat obscure, but the number and extent of small hemorrhages show that the blow must have been an extremely violent one, and it would almost seem that the brain was unable to recover from the general disintegration thereby produced. For the unilateral paralysis there seems absolutely no plausible explanation, except the pressure of the fluid, and the relief of paralysis was apparently due to relief of this pressure. The fluid would doubtless have been absorbed in any event in a few days, and it is a question whether the operation had any decided effect one way or the other upon the progress of the case. In reporting this case Dr. Brooks and myself stated that it seemed probable that the diagnosis of cerebral œdema would appear more prominently in the future than in the past as a cause of pressure symptoms, though the relation between these cases and those of so-called serous meningitis had not definitely occurred to us.

That this local accumulation of fluid may appear in other than traumatic cases is shown by a case in which Dr. J. Orne Green operated for temporo-sphenoidal abscess of aural origin. Localized convulsions and paresis appeared later in the opposite arm, giving rise to the assumption that infection had extended to the arm centre. Operation was performed at this point by Dr. Homans; an incision of this membrane was followed by a gush of clear fluid. Further exploration of the brain revealed nothing beyond an œdematous condition. In this case it would appear that some obscure vasomotor influence had produced local transudation. Possibly some obscure reflex nervous irritation determines such conditions, as suggested by Quincke in comparing local cerebral œdema with transient peripheral swellings, as of the face. It is not improbable that the temporary paralyses of general paralytics owe their origin sometimes to local serous exudation.

The practical bearing of this discussion as regards traumatic cases is upon the question of surgical interference. In two of the cases here mentioned operation was undertaken, and in the other two it was seriously considered and prepared for. The result of the operation was in both cases negative, the severity of the underlying condition in each being so great as to preclude recovery. In the other two cases absorption of fluid was apparently rapid and recovery perfect.

It is of great importance to have this class of cases in mind before operating for supposed meningeal hemorrhage. The following diagnostic suggestions were offered by Dr. Brooks and myself, based upon our experience:

1. In case of hemorrhage the unconsciousness, as a rule, after once appearing has steadily deepened.

2. The other symptoms show, as a rule, *steady* and *continuous* progression.

3. The insensibility to pain is generally profound in case of hemorrhage after unconsciousness is established.

4. Inequality of pupils is likely, though not certain to be present, in hæmorrhage.

The cases under consideration, on the other hand, have run a comparatively atypical course, and pressure over the hæmatoma has generally given rise to signs of discomfort, long after the unconsciousness has been so profound that no response to questions could be elicited. The pupils have generally been alike, though reacting sluggishly to light. Diminution or absence of knee-jerk has been a fairly constant symptom.

With regard to the question of operation, if the atypical course of the case tends to eliminate hemorrhage, I should favor delay, even though localized paralysis be present. This is especially true in children, not only because they appear particularly prone to temporary paralysis after blows upon the head, but also because operation is borne much less well in early life, and there is always the fear lest the surgical operation may precipitate a fatal issue.

Conclusions.

First. A severe blow on the head may result, either directly or by *contrecoup*, in a local bruising, congestion, and swelling of the brain-tissue, with serous exudation into the subarachnoid space, either with or without œdema of the brain substance.

Second. If this accumulation of fluid occurs over the motor centres it may be imprisoned so as to cause focal pressure symptoms, simulating meningeal hemorrhage.

Third. This accumulation of fluid is not compensatory, but represents an ineffectual effort toward relief of tension, as shown by the swollen condition of the underlying brain substance when exposed by operation. The mechanism is probably analogous to if not identical with that of the so-called serous meningitis of Quincke.

Fourth. The lesion is self-limiting, the resulting paralysis disappearing in the course of a few days.

Fifth. This condition may be mistaken for middle meningeal or middle cerebral hemorrhage. The diagnosis is difficult and sometimes impossible. Factors aiding in the diagnosis are (*a*) an atypical course, (*b*) absence of steadily increasing coma, and (*c*) the appearance of sensitiveness to pain on manipulation of the head, even after the unconsciousness is so great that questions are not answered. The general symptoms (restlessnes, stupor, headache, and moderate febrile movement) may be the same in both conditions.

Sixth. The mere presence of paralysis following a blow upon the head

is not necessarily an indication for immediate operation, and in the absence of steadily deepening unconsciousness and of steady progression of other cerebral symptoms, it will be often wise to postpone surgical interference, though generally speaking an exploratory operation is always justified in case of focal paralysis following head injury.

Seventh. This lesion is to be particularly borne in mind in the case of children and young adults, and perhaps in alcoholic patients. In elderly patients the same set of symptoms points more decidedly toward hemorrhage.

THE PATHOLOGY AND MORBID ANATOMY OF HUNTINGTON'S CHOREA, WITH REMARKS ON THE DEVELOPMENT AND TREATMENT OF THE DISEASE.

By Joseph Collins, M.D.,

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Introductory Remarks. *Point of view.* In attempts to solve the problems imposed upon the neurologist by the exigencies of his specialty, many difficulties are encountered. Of these none has the secret of its genesis and being more carefully concealed than the hereditary degenerative diseases. The pathogenesis of the acute inflammatory diseases of the nervous system is an open book. The process is essentially the same as that of acute inflammation in any other tissue, individual attributes and variations being conditioned by structural peculiarities. It is in reality the reaction of the tissues to different forms of irritation, principally bacterial. The degenerative diseases, on the other hand, yield the mystery of their being and causation in a discouragingly slow way. This is true especially of the degenerative diseases that are handed down from generation to generation, such as the hereditary ataxias, choreas, and dystrophies. Although we may apprize ourselves fully of the terminal morbid anatomy of such diseases, it is almost as impossible to infer the pathogenesis of them from a study of their morbid conditions alone as it is from a consideration of the symptoms. In other words, the status of the beginning or original lesion cannot always be inferred from a consideration of the morbid conditions found at the time of death, particularly if the disease has existed a great number of years. This statement may sound heretical, but I am convinced that it does not materially overstep the boundaries of truth. No one can spend his days in the laboratory at work on the central nervous system of individuals who have succumbed to degenerative nervous diseases of

[1] Read before the Neurological Society, November 2, 1897.

protracted duration without having forced upon him the fact that there are certain abnormalities of the circulatory system, consisting of a variable degree of degeneration of the vessels, change in the size of the lymph spaces, and relative disproportion of glia tissue to the parenchyma, which occur with all degenerative diseases, considered entirely apart from their causation. The longer I study the morbid anatomy of the degenerative nervous disease, the more I am convinced that we cannot be too chary of attributing such departures from normal, as these just enumerated, to the direct effect of the action of the factors to which the disease is attributed. On the contrary, I venture to believe that these terminal changes are very often secondary; that they are entitled to no other significance than evidences of protracted disturbance of nutrition, and that this nutritional depravity results from the functional disorder of the part or the organ as well as from the existence of the original lesion. There is nothing more certain than the occurrence of glia proliferation in all slowly progressing destructive lesions of the nervous system; but nothing can be more misleading, in my estimation, than the idea that this glia overgrowth is primary, and the changes in the parenchyma secondary. The fact that the overgrowth of glia is most frequently into the pericellular and perivascular spaces has led to the belief that it is responsible for the changes in the cells which are found accompanying such proliferations. This is a return to a theory which few pathologists accept to-day, viz., the theory advocated by Bevan Lewis that the glia cells have scavenger properties and attack diseased nerve fibres and cells.

A retrospective glance of the contributions that have been made to the morbid anatomy and pathogenesis of the hereditary degenerative disease known as Huntington's chorea prompts the foregoing remarks. The disease is comparatively rare, and of somewhat recent recognition. Consequently, the luminous contributions that have been made to its pathological history are very few. Moreover, the reports that have been made are not uniform regarding the conditions found macroscopically and microscopically. Thus, one writer contends for the genetical relationship of the diseased tissues to the vascular system; another that the beginning of the pathological process is in the cells; while others maintain that the primary manifestations of disease are in the neuroglia. These discordant views are, I venture to suggest, the resultant of varying points of view of different observers. We should bear in mind, when given opportunity to study the central nervous system of cases of Huntington's chorea, that it is scarcely justifiable to contend that all the morbid conditions found microscopically are inherent to the disease. They may or may not be. Many of them, as I have said, may be the consequence of prolonged interference with nutrition. Naturally, it must be granted that this interference with nutrition becomes a part

of the disease; but it is incidental, not essential. It follows, therefore, that study of cases of Huntington's chorea in which the anatomical material has come early to the investigator's hands through some accidental cause, such as the fatal outcome of one of the acute diseases, is of greater value in illuminating the pathogenesis of the disease than is study of cases that have died a natural death after the disease has run its full course. Although the investigation of Huntington's chorea which I present herewith was made on the nervous system of an individual who had the disease for a number of years, I shall take occasion to compare the findings and contrast them with a case reported by Dana about three years ago. It will be seen that the findings are very similar in the two cases, although the patient reported by this writer had been afflicted with the disease but a comparatively short time, and died from an attack of typhoid fever.

Together, they may contribute toward establishing the morbid anatomy of this disease and toward indicating the pathogenesis. They may, perhaps, necessitate a modification of the statements made by some modern writers on chorea, such as Osler and Blocq, to enumerate two of the most recent, who state, the first, that nothing has yet been found to explain the true nature of the disease, and the second, that it is not yet possible to say what the anatomical substratum of the affection is.

History of the Case. The history of the patient, so far as I have been able to ascertain it, is as follows:

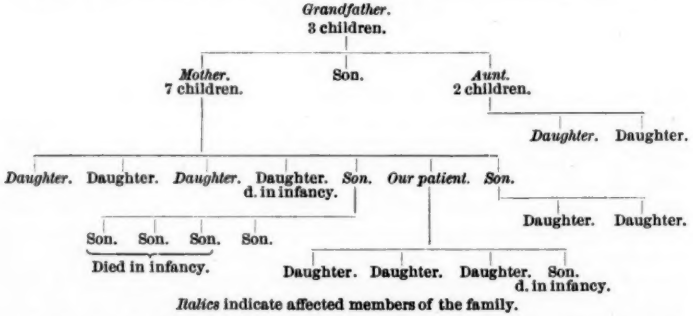
An American gentleman, aged fifty five years, married in early manhood and begot four children, three girls and one boy, the latter of which died in infancy. His wife lived but a short time after the birth of the last child, which is now ten years old. His ancestors were settlers of upper Manhattan. They had been large families, and the victims of a disease, characterized by jerking, uncontrollable, dance movements and mental decay, showing itself in early middle life, which led some of its victims to asylums and others to voluntary seclusion. The disease from which they suffered is the one now known as Huntington's chorea.

Mr. X., fully and intelligently aware of his heritage, was quite well until the forty-fourth year of his age; at least, it may be said that, although he had been very sensitive to comment or discussion concerning his health and very nervous and somewhat bizarre in his movements before that time, he was still able to transact business. From the time when the disease first showed itself in sufficiently definite form to be diagnosticated until the time of his death more than ten years elapsed. In the beginning the twitchings or choreiform movements involved only the hands, but within two years from the time of their advent they were to be seen in the cephalic extremity, and in the last years the face and tongue, and eventually the lower extremities were much affected. I have been assured that the legs were last involved, and that the gait did not show the rather typical "halting, stalking" feature that has been so often commented on by reporters of these cases. His mind remained in fairly good condition until about three years before death, when he

began to have suspicions of his family and of his friends. He was with difficulty convinced that they were not plotting to put him in an asylum and to separate him from his children. He became depressed, sometimes suicidal, very forgetful, and distrustful of those on whom he had had the greatest reliance. His most persistent concern was to prevent separation from his children. Gradually he withdrew from association with his friends in society and in business, and kept near his children. The movements of the upper extremities and face were such that his appearance on the street attracted attention, and this made him loath to go abroad. In the last years of life speech became indistinct, and for several months before death it was with difficulty that one understood him. For a few weeks before he died the movements of the face and tongue were so severe that he rarely attempted to speak, and when he did the result was a mere verbigeration.

I saw him for the first time a few days before his disease terminated. He was apparently suffering from some acute illness, although nothing could be made out from physical examination save the choreiform movements and hyperthermia. The statement of the patient's family was that latterly the choreic movements had steadily increased in intensity, and that the mind had become very much clouded. About a fortnight before his death a fever developed, which remained with him until the end. When I saw him the temperature was 105°, and thereabouts it continued until the time of death, several days later. At this time the movements, although very severe, were not sudden and bizarre, but irregular and made worse by volitional intent. They were very severe and incessant, except during profound sleep. Even then they were seemingly disturbing and the cause of frequent awakening. Although the patient was quite conscious, he made no verbal responses or requests. The movements of the face and tongue were conspicuous and severe, and the irregular twitchings of the body so profound that he had to be maintained forcibly in bed. Aside from evidences of senility, the examination showed nothing of sufficient interest to require mention. There was slight rigidity of the extremities, but there was no exaggeration of the reflexes. The cause of death seemed to be exhaustion and high temperature.

The family history is shown graphically by the following scheme:



Italics indicate affected members of the family.

Summary of the family history. In brief, illness of this nature is traceable in the patient's ancestry to the maternal grandfather, an Irishman.

Whether or not the taint could be traced further back, if access were had to the family records, it is impossible to say. Of the children of this grandfather three in number, two daughters were affected. One of these daughters was mother to our patient, and bore seven children, five of whom became afflicted with the disease. The other daughter bore two children, and of these one became choreic. In three generations there have been no less than nine members affected, and when the fact that many children of these families died in infancy is considered, the number of cases that have developed is surprisingly great. The disease has not yet manifested itself in the children of the third generation, but evidences of neuropathic inheritancy are to be seen in almost every one of them, and the children of our patient possess a number of striking somatic stigmata of degeneration.

Post-mortem findings. The notes of the autopsy, made four hours post mortem, are as follows: The body, showing slight rigor mortis, is but little emaciated. There is marked panniculus adiposus, and when the abdominal cavity is opened the organs present a healthy appearance, save that the tip of the appendix is adherent to the colon. The lungs show a few calcareous deposits and old adhesions at the apices, and one of these adhesions on the left side binds the pleura above to the mediastinum; another, below, to the diaphragm. There is a considerable deposit of subpleural fat, and both lungs are slightly emphysematous. The pericardial sac contains a customary amount of fluid; the heart is small, and, to the naked eye, presents the appearance of slight fatty infiltration. The valvular apparatus is normal, the endocardium quite smooth, and the large bloodvessels at the base of the heart are soft. The liver is of normal size, weight, and consistency; but cross-section reveals a considerable degree of fatty infiltration. Save for a slight increase in volume, the spleen is normal, both *en masse* and when cut across. The only feature worthy of remark shown by the pancreas is a rather uncommon state of the bloodvessels. The walls of the vessels are hard, the calibre is increased, and the lumen is excessively patent. The kidneys show no departure from normal, with the exception of an ancient and rather well-marked scar at the upper end of the left kidney. The capsule is easily stripped, and when the kidneys are laid open the markings of the cortical and medullary parts are very indistinct, and there is marked venous congestion. All the visceral organs are richly embedded in fat. The intestines are quite normal, and nothing whatever is found to account for the hyperthermia which the patient had, or to point the cause of death.

On opening the skull the dura was considerably adherent, the diploë dense, and the Pacchionian depressions well marked. In the lower end of the longitudinal sinus there was a small coagulum. There were a number of adhesions between the pia and the dura over both hemispheres, more on the left than on the right. These areas of adhesion are not at all firm. There is slight excess of cerebro-spinal fluid, and the brain has a "wet" appearance. The pia is not adherent on the internal surface, but while it is in contact with the brain it has a congested, thickened appearance. This is particularly noticeable over the pons, where the arborization is striking; but when the pia is stripped, it has its customary appearance. The convolutions of the anterior pole of the brain seem very small, and the central convolutions are strikingly narrow. The weight of the entire encephalon was forty-three and one-half ounces.

When the spinal canal was opened considerable fluid was found in the cerebro-spinal space, and after incision of the membranes more than three ounces of this fluid was collected. The cord presented a "wet" appearance like the brain. The most remarkable feature of the meninges of the cord was that the dura was intimately adherent to the spinal column. Instead of hanging loosely, as it does naturally, it is adherent throughout its entire length, so that the dura and the posterior spinal ligament are one.

Abnormalities of the brain, as indicated by measurements. Inspired by the careful and exact study of the brain made by Dr. Charles L. Dana in the case of Huntington's chorea reported by him, I made not only a close examination of the brain for fissural and convolutional anomalies, but also a large number of measurements to determine the relative thickness of the gray matter of the cortex. The first awarded nought, as there seemed to be less evidence of variation than it is customary to find in routine examination of brains. The principal fissures were wider, shallower, and shorter than they are normally, and this is particularly noticeable in the central fissure, which is but a trifle more than ten centimetres in length. But there is nothing that points to defective evolution in the unfolding of the brain. Perhaps the most conspicuous feature of the brain in the fresh state is a cribriform or sieve-like appearance of the cortical and subcortical substance, to be seen very strikingly on cross section of the Rolandic region, and a peculiar mottled or tier-appearance of the cut brain substance. The brain seemed excessively vascular, both *en masse* and when cut into, but there were no foci of hemorrhage, and the vessels on the surface were in a good state of preservation.

The frontal convolutions and the motor areas of the brain were taken for special study. The anterior and posterior Rolandic gyri, in length ten centimetres, were divided into three parts. The first portion, that is, the portion bordering the longitudinal fissure, was cut into three pieces, and the first put in absolute alcohol, in preparation for Nissl's stain; the second in osmic acid, for the Golgi method; and the third in Müller's fluid, for the reception of Weigert's and Van Gieson's stains. The second third of the motor convolutions was treated in the same way, and likewise the last third, the inferior end. The motor gyri of the left hemisphere were disposed of in a similar way except that No. 1 was put in osmic acid, No. 2 in Müller's fluid, and No. 3 in alcohol. The remainder of the brain was hardened *en masse* in Müller's fluid, save a few pieces taken from the different lobes, which were put in alcohol. After the hardening process was complete ten measurements were made of the cortex of the surface from the different lobes. The averages of these measurements are here given.

	Centimetres.		Centimetres.
First frontal . .	2.60	Inferior parietal . .	2.88
Second frontal . .	2.55	First temporal . .	2.90
Third frontal . .	2.55	Second temporal . .	2.95
Ascending frontal .	2.46	Cuneus	2.35
Ascending parietal .	2.48	Precuneus . . .	2.48
Superior parietal . .	2.82	Island of Reil . .	3.60

Contrasting these averages with what has been determined to be the average thickness of the gray matter, we see that they are uniformly

less than the normal cortical gray. I do not forget that the individual whose brain we are studying was of an age when occasionally senility makes itself manifest in the brain, but the pronounced thinness found here cannot be attributed to age. Comparison with the result of measurements in Dana's case,[1] and in the more recent one of J. M. Clarke,[2] shows that the cortex was somewhat thinner than in the former's patient and more nearly approaching Clarke's case.

RÉSUMÉ OF THE MICROSCOPICAL FINDINGS. I shall preface what I have to say concerning the changes that were detected microscopically by the statement that, although they were not found exclusively in the Rolandic region and in the adjacent frontal gyri, the disease process was incomparably more advanced and conspicuous in these regions, more especially in the former, than in any other part of the brain. I shall not give a detailed account of the slightly varying conditions to be found in each specimen, but rather a *résumé* of the findings in each of the differently handled pieces. I am unwilling to say that specimens from one hemisphere show any pathological changes which would serve to differentiate them from specimens taken from the other hemisphere.

The specimens that were stained with methylene-blue, according to the Nissl method,[3] reveal more clearly than any of the other stains the abnormalities of the cortical cells, although the differentiation in cell constitution is not well-marked, and one cannot see any actual change in the chromatic structure. The most conspicuous feature is the pigmentation and shrinkage of the cells, particularly those constituting the layer of large pyramids. (Fig. 1.) The cells just beneath this layer, the so-called polymorphous cells, are pigmented to a lesser degree, but still conspicuously. The next most striking abnormality is the size and shape of the cells of these two layers. The cell processes are attenuated, spiral, and shrunken. The cell-bodies are uniformly rounded, or slightly irregular, and many of them are not much greater than one-half the normal size. (Fig. 2.) All the cells of any given area are not affected, and many of them are diseased in different degrees. The cellular implication seems to be in tiers or streaks, and to this was due the mottled appearance of the cortex observable in the fresh brain. The fact that this mottling was noted when the brain was first examined points conclusively to the fact that the shrinkage of the cells was not incidental to the process of preparation to which the tissue was subjected in hardening and staining. The pericellular spaces appear very much larger than normal, but this increase in size is more relative than actual. In the pericellular spaces there is a remarkable accumulation of neuroglia nuclei seen spinning themselves out over the body of the pigmented atrophied cell, or clinging to the outer coat of a bloodvessel. (Fig. 3.) This manifestation of proliferation of glia nuclei is more evident in the deeper layers of the cortex than in those of the superficies,

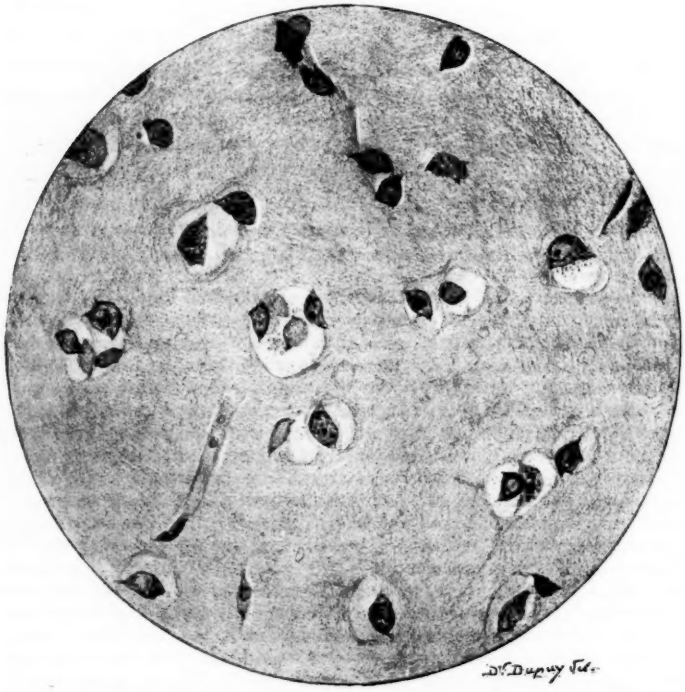
[1] Journal of Nervous and Mental Diseases, 1894, p. 565.

[2] Brain, 1887, p. 26.

[3] In the preparation of the methylene-blue sections I received much highly valued assistance from my friend, Dr. B. Onuf, and I wish to express my appreciation of his courtesy and good offices.

and particularly in the polymorphous cells and in the layer of large pyramids. The bloodvessels in specimens made with this stain are thickened, principally the result of proliferation of the nuclei of the intima. The evidences of vascular degeneration are slight, and the change in the bloodvessels is not comparable to the change in the parenchyma about it. These lesions of the gray matter were found throughout the Rolandic region, some sections showing them more prominently than others.

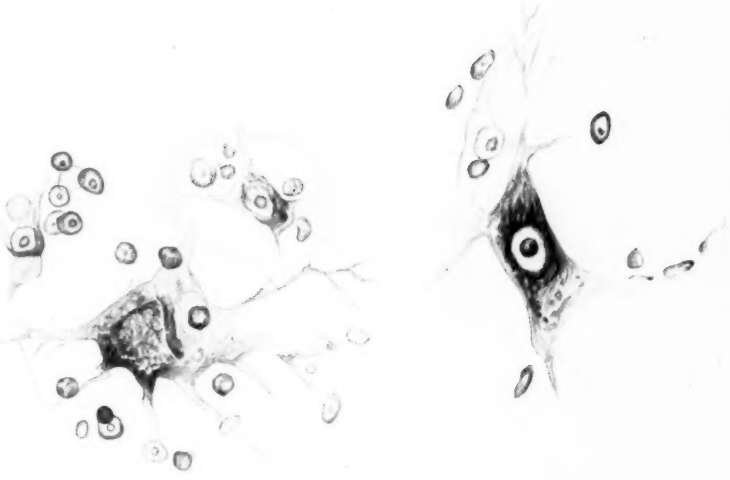
FIG. 1.



Pigmentation and shrinkage of the cortical cells and enlarged pericellular spaces.

Specimens stained with carminate of soda and with picric acid fuchsin showed the thinness of the cortical mantle which had been remarked in the fresh brain. The pia presented a comparatively normal appearance —possibly a slight degree of thickening. The most conspicuous feature of the specimens and one that immediately arrests the eye is a cribriform state of the tissue. (Fig. 4.) This is rather uniformly distributed throughout the specimens, but more marked in the inferior cortical layers, and it is due to three factors: (1) Shrunken ganglion cells; (2) enlarged and empty pericellular spaces; and (3) distended capillaries, arterioles, and perivascular spaces. The relative importance of each of these factors in contributing to the cribriform state is indicated by the

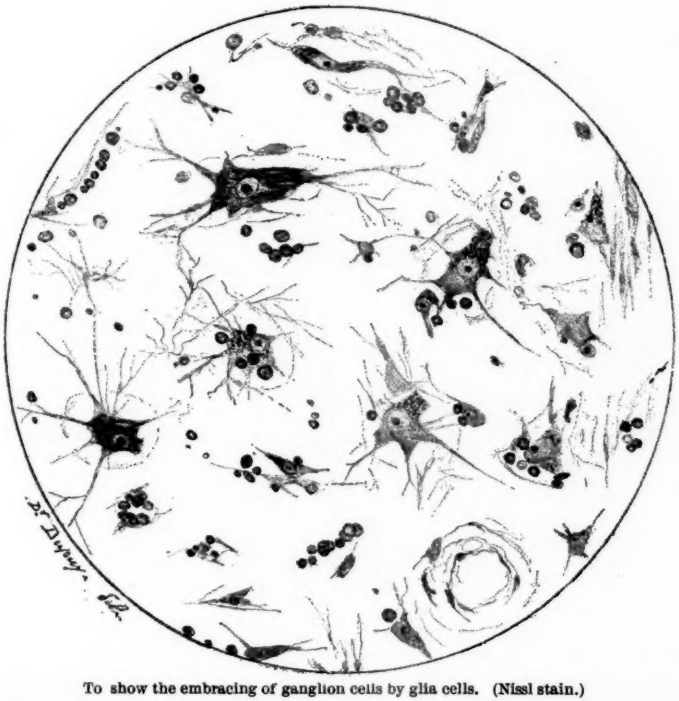
FIG. 2.



Cells from the layer of large pyramids, showing pigmentation, degeneration and pericellular distention. (Nissl stain.)

order of enumeration. When we study these three conditions individually we find that the departures from normal within the cell itself are practically the same as were determined by the methylene-blue stain. All the layers of the cortex are affected, but mainly the deeper layers, and the involvement is not uniform—*i. e.*, the same disposition to involvement in streaks already noted is here evident. Moreover, this streaky appearance is heightened by the fact that in the strands where the atrophied cells are most conspicuous many cells have completely disappeared. This stain does not permit a study of the intimate con-

FIG. 3.



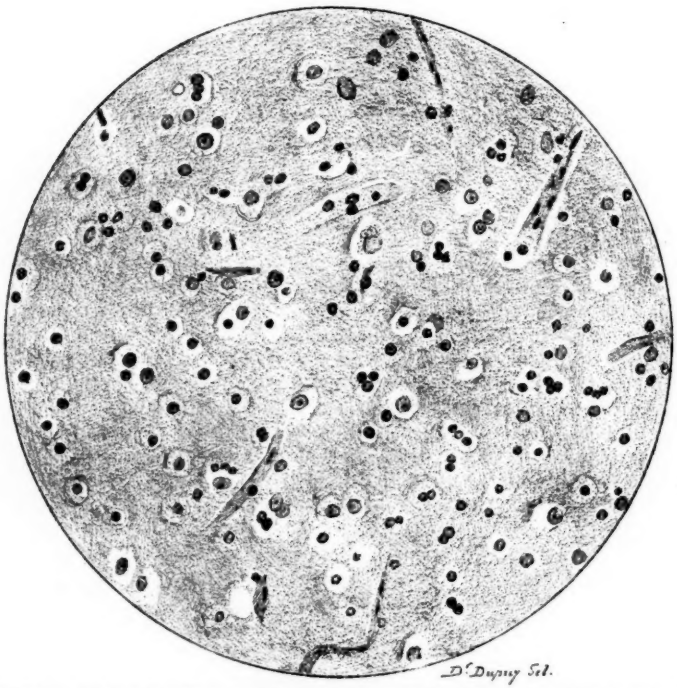
To show the embracing of ganglion cells by glia cells. (Nissl stain.)

stitution of the cell save in its reaction to the coloring agent. Many of the cells take the stain very incompletely, while others take it in an irregular fashion, which gives the cell a granular appearance. The embracing of cells by glia prolongations is to be seen with this stain much less distinctly than with the methylene-blue and silver stains. Although the calibre of many of the bloodvessels is enlarged and the lumen of increased patency, these conditions are more noticeable in the white matter than in the gray. In the latter the thickening of the vessels has caused a lessening of the calibre in one portion and an irregular bulging in another. There is nothing approaching excessive

vascularity or perivasculitis. The increase of neuroglia tissue is general, and the glia cells and fibres have accumulated in the pericellular spaces, and here and there they are to be seen on the walls of the vessels.

The most striking feature of specimens stained with hæmatoxylin are thinness of the cortex and the delicacy with which the medullated components of the cortex color.

FIG. 4.



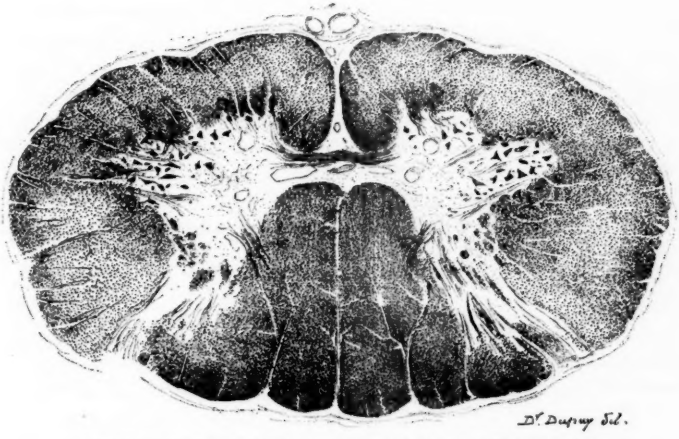
To show atrophy of the cells and cribriform state of the cortical tissue. (Van Gieson's stain.)

Specimens prepared after the Golgi method and after Berkeley's modification of it were not, on the whole, as satisfactory as the others. The greatest service that this method of staining rendered in this case was to corroborate that which had been determined by other methods. That is, it showed that the cells were not uniformly diseased; that normal, dead and dying cells were to be seen; and that the layer of large pyramids and the polymorphous cells were most diseased. The affected cells revealed their shrunken appearance and irregular outline most prominently in the deeper layers. The processes themselves, stunted and thin except where node-like enlargements have increased their calibre, are evidently much altered, especially the dendrites. Most of the dendritic

processes are quite devoid of thorns, and their inter-ramification seems less intricate and complex. The abundance of neuroglia tissue is very manifest in the preparations made with this stain, and it showed the same environmental proclivities as the specimens made with methylene-blue.

What has been said applies to the Rolandic region of the cortex. I desire to emphasize that the pathological conditions were not limited to this area, but were found to a certain degree throughout the entire brain. Sections made from the occipital and the parietal lobes revealed changes in the ganglion cells and in the bloodvessels and their surroundings which are comparable to those described in the Rolandic region, the difference being one of degree. In the frontal gyri certain specimens show the atrophic changes in the cells and the enlarged size of the perivascular spaces nearly as conspicuously as those taken from the motor areas, but the lesion is less widely distributed there, and is less advanced. In other parts of the brain there are comparatively slight changes in the parenchyma and in the supporting tissues.

FIG. 5.



Showing degeneration of the crossed pyramidal tracts. (Weigert stain.)

Examination of the pons and the oblongata does not reveal any noteworthy departures from normal. When we pass the motor decussation and get into the cervical cord there begin to be evidences of degeneration of some of the axis-cylinders of the pyramidal tracts. This becomes more apparent as we examine sections from lower levels in the cord. In none of the specimens is it conspicuously evident (Fig. 5), but there can be no question, I think, of the actual loss of neuraxons. There is comparatively slight increase of connective and neuroglia tissue, and the bloodvessels are not abnormal for an individual fifty years old. The pia is not thickened, the perivascular and pericellular spaces are well preserved, and, in short, aside from the degeneration just mentioned, the cord appears quite normal.

Summary of the Macroscopical and Microscopical Changes. If we attempt to summarize the changes found in the central nervous system of this individual with hereditary chorea, we may say that they consist, macroscopically, of:

1. Thinness and atrophy of the cortex, evidenced by the diminution of weight, by the shallowness and breadth of the fissure, by the narrowness of the gyri, and by the thinness of the cortical mantle.

2. A mottled, tier-like, or streaked appearance and a cribriform state on cross-section of the brain in the fresh state. This streaked and cribriform appearance was due to a diminution in number and in size of the ganglion cells, particularly those of the deeper layers, to an increase in size of the perivascular and pericellular spaces, and to increased patency of the bloodvessels.

The changes determined by microscopical study may be summarized as follows:

1. A decay or slowly-progressive degeneration of the ganglion cells of the cortex throughout the brain, involving principally the layers of large pyramids and polymorphous cells, particularly evident in the Rolandic region, very much less so in the anterior pole of the brain, and incomparably less so in the posterior pole of the brain.

2. Increase of glia tissue, most marked in those sections that show greatest cell implication, but nowhere sufficiently prominent to constitute sclerosis. The increase of glia tissue, although rather universally distributed, is most conspicuous about bloodvessels and ganglion cells.

3. Enlargement of the pericellular spaces and distention of the perivascular spaces.

4. Slightly diseased bloodvessels, the principal change being a proliferation of the nuclei of the tunica externa and a thickening of the tunica intima. This implication of the vessels, which is neither regular nor symmetrical, is evidenced to a greater degree in one section than in another. No punctate hemorrhages were observed.

5. Relative paucity of the medullated constituents of the cortex.

6. Slight degeneration of the crossed pyramidals tracts of the spinal cord.

To summarize the microscopical finding in a few words, I may say that the lesion is a chronic parenchymatous degeneration of the cortex, principally of the Rolandic cortex, with consecutive and secondary changes in the interstices and vascular system.

Anatomical Findings Contrasted with those of Some Recent Writers. To bring these findings into contrast with those determined by Dana, by Oppenheim,[1] and by Clarke, I shall cite a summary of the microscopical examinations as given by these three authors. In Dana's

[1] Centralblatt für innere Medicin, 1894, p 918.

case the brunt of the process fell upon the central convolutions; they suffered first and most intensely. The process was not evenly distributed, but occurred in patches throughout the affected parts. There was nothing in the intricate nature of the process to justify the opinion that it is in any sense an inflammation. There was no proliferation of connective-tissue cells, no exudation, no accumulation of leucocytes about the bloodvessels, and only a slight amount about the nerve cells. The process corresponded in every respect to one of decay and degeneration, the nerve cell itself being the first that begins to die.

Oppenheim was led, from a careful study of two cases, one a man, aged seventy-five years, in whom the disease had lasted sixteen years, and another a woman, aged fifty-six years, in whom the disease had lasted but five years, to the statement that the essential morbid condition is a miliary, disseminated, cortical and subcortical encephalitis, eventuating in atrophy of the cortical mantle. The principal changes in the brain were narrowing of the gyri, broadening of the sulci, especially in the motor, parietal, and occipital regions, deficiency in number and size of the pyramidal cells constituting the layer of small pyramids, and minute foci of hemorrhagic infiltration in the subcortical layer.

Clarke sums up the morbid changes which he found in a case of this disease of unknown duration in the following words: "The morbid change consisted in a widespread, but partial degeneration of the cells of the cerebral cortex, especially the cells of the second and third layer, most marked in the frontal and motor convolutions, together with an increased amount of interstitial tissue and number of neuroglia cells." He adds that there seems to be no doubt that the cerebral cortex, especially of the motor convolutions, is the seat of the disease, and that his own sections go to show that the primary lesion is a degeneration of the nerve cells, with a concomitant increase of neuroglia.

It seems unnecessary to dwell upon the fact that these observations are practically in accord in pointing that the disease is essentially of the parenchyma. The slightly discordant conditions found in these cases and in my own may be legitimately accounted for by a consideration of the ages of the patients and the duration of their symptoms. In Oppenheim's case alone were there punctate hemorrhages. This should cause no astonishment, when we recall that his first patient was a very senile individual who died of cerebral apoplexy, and that his second patient succumbed to an acute infectious disease, viz., influenza. In both instances the bloodvessels were enormously disposed to rupture, the first from the advanced age of the patient, and in the second because of the infection. No importance whatever should be attached to these hemorrhages, except that they were accidental. The minute vessels of the brain in the case that I studied were fittingly and quite prepared for rupture, and I have no doubt that this would have occurred if the

patient had lived sufficiently long, or if he had suffered from some acute disease, although naturally I hasten to remark that the occurrence of punctate hemorrhages, even under such auspices, would be by no means sure to follow.

Dana's findings I regard as of signal value, first because they duplicate in every respect those of my case, and second because they were found in the brain of a patient who had had the disease but a comparatively short time. The results of the microscopical study in both of these cases point unequivocally to the fact that the primary lesion is in the cell, and that the change in the cellular structure conditions not alone the phenomena of the disease, but the concomitant changes in the brain and spinal cord.

The most determined opposition to this view comes from Kronthal and Kalischer; but a careful examination of their writings on this subject convinces me that the contradictory evidence furnished by them is more apparent than real. They examined the central nervous system in a large number of cases of chronic chorea, and from their own experience, and from a consideration of the findings of others, they concluded that Huntington's chorea is in reality a diffuse, seldom circumscribed change of the brain cortex, consisting essentially of disease of the bloodvessels, which leads not infrequently through proliferation of nuclei and cellular hyperplasia to punctate hemorrhages and of proliferation of the glia and interstitial tissue. The nervous elements themselves are comparatively unaffected. Aside from the fact that the occurrence of such changes in Huntington's chorea is completely disproved by their absence in three of the most recent and most carefully studied cases of the disease (those of Dana, of Clarke, and myself), there are a number of arguments that may be brought forward to controvert this view. In the first place, and personally I consider this paramount to all others: it is not in accord with the tenets of pathology to consider together, except for purpose of contrast, or to attempt to parallelize the anatomical findings in cases of chronic Sydenham's chorea with those of Huntington's chorea. The first is an acute disease, in all probability dependent upon a specific cause, most likely an infection. The exciting factors, whatever they may be, exercise their pernicious activity upon the anterior half of the brain, and thus condition the symptoms of the disease. No doubt they may cause, if severe and continuous, real, definite, anatomical changes, and such changes may be demonstrated after death. But when the brain shows such a lesion all the tissues of the part are affected—the bloodvessels and the parenchyma, principally the former. When such conditions become chronic the characteristic lesion of a diffused or a circumscribed, hemorrhagic or non-hemorrhagic encephalitis ought to be found. Huntington"s chorea, on the other hand, is a chronic disease from the beginning. If there is any one disease be-

yond being shelved with a pathogenic microbe, or with non-specific infection of any sort, this is the one. The most reliable evidence seems to me to point to the fact that it is a disease not dependent upon any cause acting through the bloodvessels or upon any change in the bloodvessels. This is in conformity with the admitted pathogenesis of the chronic degenerative nervous diseases. In every one of these diseases, without exception, there is some implication of the bloodvessels; but if disease of the bloodvessels were responsible for the occurrence of the degeneration in the parenchyma, then these disorders would needs be incomparably more frequent in association with disease of the arterial system than they really are. Nor do I believe that the increase of glia substance in any way points to the existence of a slowly developing vascular lesion as the primary condition. More than twenty years ago Weigert established the principle that diminished mutual resistance between tissues is followed by a process of proliferation of neuroglia cells, and the presence of abnormally developed glia tissue in the case such as I have reported indicates merely such perversion of resistance.

I am reluctant to say in so many words that Huntington's chorea is a disease dependent upon a slowly progressive decay of the parenchyma —the ganglion cells of the brain, more particularly of the anterior half of the brain, the Rolandic and the frontal regions—but the bulk of evidence bearing on this subject indicates so clearly that this is a fact that such a statement seems not alone warrantable, but is demanded. I wish particularly not to attach too much importance to any one terminal change or set of changes, such, for instance, as the change in the cells in this case. But when it is kept in mind that these abnormalities have been found by several observers in cases that terminated at very different stages of the disease, one is almost forced to the conclusion that this is the anatomical substratum of the disease.

A Word Concerning the Genesis of the Disease. That Huntington's chorea is a disease conditioned by pre-natal factors, no one can deny. Yet I cannot, on this account, believe with Dr. Dana, that it belongs to teratology, using that word in the strict sense of its derivation and application. The fact that the germ plasm of the ancestry is freighted with the burden of death twenty or thirty years before the usual time does not warrant me in considering the disease either a congenital malformation or a monstrosity. It is highly probable that the proton of the ganglion cells may be properly formed, and, indeed, it does not seem to me likely that the cells of the Rolandic cortex if examined during the years of a patient's health would reveal any departures from the normal. In other words, it seems to me that we may infer a change in the germ plasm, a predisposition to early death, without a change in the germ cell. To express the nativity of Huntington's chorea more colloquially, it may be said that the ganglion cell

whose function it is to subserve motion and give basis to intellectuality is genetically lacking in the power which will enable it to exist as long under the same environment as the ordinary ganglion cell. This idea is in strictest conformity with Weismann's theory of heredity.

Remarks on Treatment. I should hesitate to allude to this sad phase of this subject did I not feel compelled to do so from a sense of duty. Not that I am able to suggest anything that may be of service, even in the amelioration of these unfortunates' conditions after the disease has developed; but I desire to emphasize the laudability of endeavoring to delay the advent of the disease in those to whom it is a natural heritage, and to add my testimony to the utter futility and ridiculousness of tenotomy of the eye muscles, graduated, non-graduated, or any other kind, advocated by therapeutic pretenders. I am prompted to this remark by the fact that the most recent member of this family to show manifestations of the disease is now under the care of an individual whose understanding of the pathogeny of chronic degenerative nervous diseases of the convulsive order prompts him to the promise of a cure in such a patient as this. If one can look in the microscope at the sections from the Rolandic area of the cortex, such as those I have shown, and then state that such a condition can be overcome by any procedures directed toward the eye-muscles, he is a type of mind that surpasseth all understanding.

I shall take the liberty of making one therapeutic suggestion apart from those usually given in systematic writings on this disease, and shall preface it by saying that I do not forget the necessity of preventive, hygienic, and therapeutic measures of any kind that have in the past been shown to be of the slightest service in either delaying the disease or ameliorating its symptoms. The suggestion that I desire to make is this: All chronic nervous degenerative diseases, it matters not whether they be of the brain or spinal cord, be they convulsive or paralytic in manifestation, are benefited, if at all, by toxic substances given in toxic doses. We know not how they act; we know only, as a matter of clinical experience, that to secure the efficacy of the bromides or of opium in epilepsy, it is necessary to give them in poisonous doses; that strychnine may be of any service in progressive muscular atrophy, or that nitrate of silver should stay the process of locomotor ataxia, they must needs be given in doses to make their poisonous symptoms manifest. Ordinary medicinal doses of these drugs directed to the cure of the disease for which they have been mentioned are practically useless. Therefore it has occurred to me that if we wish to influence the course of hereditary chorea after it has once made its existence manifest, it is necessary to administer the elected substance, be it arsenic, opium, serum, or vital extract which it is hoped may be of service, in the largest possible doses consistent with life, and to maintain such administration for a prolonged time.

If experimental therapeutics would but teach us the changes that go on in ganglion cells as the result of administration of such poisons, revealable by the methylene-blue method, we would be started on the road to the discovery of some measure for the radical cure and treatment of many diseases now treated empirically. Such studies have already been begun, and no one can allow his thoughts to dwell upon the morbid anatomy of chronic nervous diseases without indulging the hope that they may be continued.

Although the physician is not a deputized nor a self-constituted monitor of society, nor should he even have a desire so to be, nevertheless he may legitimately indulge himself in following the dictates of altruistic sentiments for society's welfare. In the opinion of the present writer he cannot more wholesomely or adequately give voice to an expression of these aims than by using such influence as he may possess against marriage for those whose heritage is such a disease as Huntington's chorea, and who, in case progeny be vouchsafed them, will undoubtedly propagate it, no matter how supremely vital the germ plasm of the other parent may be.

TRAUMATIC NERVOUS AFFECTIONS.

A STUDY OF TEN CASES.

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In attempting to study ten cases of nervous affections following violent injuries, only the more salient features of their histories will be given. The literature of this subject, which is now very voluminous, has been so recently gone over by several writers[1] that I will refrain from any similar effort in this paper.

As is well known, not only vague, fleeting, and indefinite nervous symptoms follow and result from traumatisms, but many definite nervous affections as well. On the face of the subject, it would seem absurd to suppose that any violent general shake-up of the body would produce one particular nervous affection; for, in general, we know that traumatisms, like other existing causes of disease, produce various affections depending upon the predisposition and temperament of the individual. Of a number of persons subjected to what seems to be the same sort of traumatism, some may suffer from severe nervous symptoms while others may escape such sequelæ. This, again, is to be explained

[1] Dana (Hamilton's System of Legal Medicine, p. 297) and Knapp (Nervous Diseases, by American authors, p. 135).

on the theory of difference in predisposition and resisting powers, although it is well to bear in mind that it is exceedingly difficult to say of any two cases that the trauma was of equal or approximately equal severity. In view of these facts it would seem unlikely that violent traumata would set up a particular sort of nervous affection, a disease entity, to which a specific name, such as "railroad spine," "traumatic neurosis," or "spinal concussion," could be applied; but it must be admitted that certain of the nervous symptoms following these violent injuries are much the same. It is this fact, indeed, that has led many to describe these affections as though they belonged to a single disease entity. But the pathology is obscure, and, no doubt, varies greatly. Post-mortem records are scant, indeed. To be sure, such lesions as hemorrhage and lacerations have been found, and symptoms arising from these are fairly well recognized, but of cases with a vague and indefinite symptomatology, the pathology may be said to be practically unknown. For this reason it is not logical to say, as some have, that all these nervous cases get well; no more than the same could be said of hysteria or neurasthenia. The cases to be studied in this paper will be considered from the clinical and medico-legal aspects, for I have no autopsies to offer. In eight of these cases damages were claimed, while in the two remaining cases, in which no such claims were made, the symptoms were more severe and persistent than in some of the other cases. Although damage claims were settled in one of these cases some months ago, no improvement followed.

These ten cases may then, from a medico-legal point of view, be divided into two groups, the first of which will comprise eight cases in which damages were claimed, and the second group two cases in which no damages were claimed. The question of the genuineness of the subjective symptoms in these nervous affections following violent accidents has long been a bone of contention, and although it has been very generally admitted that the element of exaggeration plays an important *rôle* in many of them, there are to-day only a few neurologists who contend that simulation enters into them largely. I know of no method of clinical study which is so well calculated to throw light on these mooted points of exaggeration and simulation in this class of cases as such a comparison between litigation and non-litigation cases; and while the cases to be reported will be considered with respect to this point chiefly, yet there are certain points relating to symptomatology, prognosis, and treatment which will also be considered.

Seven of these eight cases comprising the group of litigation cases received their injuries through an explosion of natural gas, which occurred February 27, 1895. The seven persons injured in this accident were all at work in a cigar-factory when the explosion occurred. The building was greatly damaged, and most of the seven persons who

afterward claimed damages received surgical injuries concerning which there was no controversy. These seven cases will be first related.

They were all examined by Dr. J. P. Shaw and myself in March, 1896, a little more than a year after the accident. But Case I. had also been examined by us previously—on May 29, 1895. To describe this first examination of Case I., I will draw from the notes and report Dr. Shaw and I made at that time:

Case I.—Man, aged thirty-seven years. *Personal history* (as given by litigant and his physicians). He has been married eight years, and is by occupation a cigarmaker. He has always enjoyed good health, and has worked uninterruptedly at his trade (cigarmaking) for the past fourteen years, save for an attack of typhoid fever, from which he suffered thirteen years ago, and from which he fully recovered. His weight for the past ten years has ranged from 135 to 145 pounds, generally staying at about 139 pounds. Since the accident to be described he has been run down in flesh, and now weighs 123 pounds. On February 27, 1895, while at work in the second story of the cigar-shop, a gas explosion occurred which projected him up in the air from the table at which he was at work. He alighted face downward, striking the left testicle in doing so. He was unconscious for a few moments. Returning to consciousness, he immediately arose and knocked out a window, and went around on the outside of the window-sill to another window, and there assisted from the building some girls employed in the factory. He then got his coat, and, ten or fifteen minutes later, proceeded to the office of his physician. About this time he noticed that he was bleeding about the mouth, and that the canine and first bicuspid teeth of the right upper jaw were missing. Immediately after the accident he noticed swelling and pain in the left testicle. He noted also a large black-and-blue mark in the left groin. He experienced nausea and vomited. Upon retiring that night he noticed that the swelling in the testicle had increased; he also noted slight tenderness over right parietal region, just to the right of median line, but paid no attention to it, and stated to us it did not amount to anything; the left testicle continued to swell through the two following days, when it had attained its height, and remained so swollen for another week, when it began to subside, and has continued to do so up to the present time (May 29, 1895). Three days after the accident he took the position of watchman about the building in which the explosion had occurred, and continued in this capacity for two weeks, suffering all the while from some pain arising from the swollen left testicle, and at times from nausea, but complaining of no other trouble. After acting as watchman for two weeks, he was one day seized with weakness and pain in his head (in median line, three inches anterior to the occipital protuberance), and walked home without assistance, and told his wife he was sick. From this time his mind was a blank for a week and a half, at the end of which period he returned to consciousness to find he had lost some power in the left side of the body. His physician states that during the period he was unconscious urine dribbled away from him, latterly most of the time, being tinged with blood. This hæmaturia continued until May 27, 1895, or two days before this examination. The return to consciousness was gradual, taking place in two days.

Examination. May 20, 1895. The litigant is five feet six inches tall, thin, emaciated. In walking he distinctly drags the right leg. His right arm hangs rather limp at his side. He complains of pain along a line running from median line of head about three inches anterior to the occipital protuberance down to end of lower lumbar spine, and also of pain over the entire right side of the trunk, especially over the summit of the shoulder. This pain he states is present night and day. Moderately firm pressure over the spinous processes, he said, caused pain, which was excessive over the seventh dorsal, and at a spot over the dorso-lumbar junction. He states that the entire side of his body was insensible to pain. We tested this by pinching, and by plunging needles into the skin at various points over this region and by applying a very strong faradic current. Both these procedures, he stated, were painless. Deep pressure over the right side caused pain, especially over the upper portion of the right trunk. Tactile sense was, as shown by his answers, greatly diminished over the entire right side. It was noted in testing that deep pricks with the needle brought no blood, and that the gaping cavity produced by the pricks seemed wax-like. The muscular power of his right leg and arm was apparently diminished. With the dynamometer the grasp of the right hand showed eight pounds; tests with the galvanic and faradic currents showed the presence of neither quantitative nor qualitative changes in muscular responses.

The left shoulder was distinctly higher than the right. There was some slight scoliosis in the dorsal region (to the left). The penis and testicles appeared normal. There was a slight abrasion over the left pubic region (probably caused by bandage). Deep pressure over the left perineal region, he said, caused great pain.

Abdominal and cremaster reflexes both active. Knee-jerks: right, present but diminished; left, present, normal or slightly exaggerated.

Fields of vision: Right, greatly contracted; left is also distinctly contracted (both for white).

Pupils react to light and accommodation.

Urine: specific gravity, 1020; acid; no albumin; no sugar. Microscope shows oxalate of lime crystals and amorphous phosphates. No blood, pus, or epithelial cells.

COMMENTS. His thin, emaciated appearance would impress any one that he is ill. His manner and general bearing lead us to believe that he is honest in his various assertions. We detected no efforts at malingering or concealment. That the accident caused Mr. H. to lose two teeth and to injure his left testicle so that it became swollen and inflamed, we assume can be taken for granted. The hæmaturia may have been caused by some injury to the prostate or deep urethra or bladder, but it would seem that whatever damage the testicle and urniary apparatus may have sustained has now been largely repaired. There remain for consideration the symptoms of coma, pain, general weakness, emaciation, hemianæsthesia, and hemiplegia. These demand serious consideration. We are of the opinion that these symptoms are very largely or wholly produced by the nervous state of the litigant, which we believe to be one representing an admixture of neurasthenia (nervous exhaus-

tion) and hysteria. The reasons favoring this view are twofold, viz., negative and positive. The appearance of hemiplegia *some days after this accident* and its mode of onset are against organic disease being the cause of it. It is well known that hemiplegia is almost invariably of cerebral origin. Spinal lesions are very generally the cause of paraplegia. In Mr. H.'s case, the history of head injury to account for this hemiplegia is entirely wanting. If a gross spinal lesion were present he would, in all probability, if paralyzed at all, be symmetrically paralyzed. On the other hand, partial right hemiplegia, with complete anæsthesia of the paralyzed side, and great loss of tactile sense without facial paralysis, without aphasia; the spinal tenderness, with two points of especial tenderness (hysterogenic points); the markedly contracted visual fields; the failure of blood to follow needle pricks, and the gaping cavities left by the same, all very strongly point to the right-sided paralysis and loss of sensation as being of hysterical origin. Spinal tenderness is often seen in cases of neurasthenia. The man's generally weakened condition may most probably be attributed to nervous exhaustion.

Medical literature shows that hysteria and neurasthenia frequently follow such accidents as the one from which Mr. H. suffered, and it is, moreover, well known that fright is one of the chief causes of hysteria. Following the accident there was, very likely, prostration, relaxation, and irritability of the nervous system, and this constitutes the neurasthenic element in the case.

In March, 1896, ten months after the above examination and report were made, Dr. Shaw and I again examined Mr. H. We noted that he walked better, although his gait was different from that observed at our first examination; he walked now by raising up the entire left side through the action of his trunk and pelvic muscles. The pain in the perineum was less; the knee-jerks were unchanged. His weight was ten pounds less than at the time of our first examination; the right-sided anæsthesia, involving conjunctiva and buccal mucous membrane, was still absolute. The spots of spinal tenderness remained; there was no atrophy. He stated that he was able to only partially open his mouth, and that he had been troubled in this way for the past six or seven months. At short intervals he closes his eyes firmly. He stated that his appetite was good and that he slept well. On the whole, there was little change noted at this examination.

Remarks. What has been already said as to the nature and genuineness of the symptoms in this case needed, in my opinion, no alteration after this second examination. That the partial hemiplegia following a coma which came on seventeen days after the accident, absolute hemianæsthesia, spinal tenderness, and restriction of the visual fields could be simulated, I regard as so highly improbable that the supposition is, in my opinion, preposterous. The prognosis at our second examination

seemed more grave than at our first examination. Damage claims had not yet been adjusted.

CASE II.—A girl, aged twenty-four years. She received a number of bruises and was very nervous and excited after the explosion. A lump the size of the fist raised on the back of her head. The swelling and bruises disappeared in the course of a month, during which time she was confined to bed. Since the accident and up to the present time she has complained of almost constant pain in the back and neck, and has been subject to frequent headaches. She states that she has less strength and that her power of endurance is greatly lessened. There is marked tenderness on pressure over fifth, sixth, and seventh thoracic vertebræ. She states that for three or four months following the accident she slept poorly, but that she has since slept well. Her general appearance is excellent.

REMARKS. The nervous symptoms of this case were entirely subjective. But we believed they were genuine, (1) because we learned that they were constantly complained of, (2) because they are symptoms ordinarily seen in neurasthenia, and (3) because it seems to us highly improbable that such a train of symptoms could be simulated, and for so long a time.

CASE III.—D., girl, aged twenty-one years. Her conjunctivæ were inflamed after the explosion. For three weeks following it she had frequent, violent laughing spells. Ever since the accident she states that she has slept poorly. Frequently she wakens in the night, and often has terrifying dreams. Her strength and endurance have been much less since the accident. She has headache almost constantly. There are points of tenderness over the first, sixth, and tenth thoracic spinous processes and at lumbo-sacral junction. There is a small spot of anæsthesia about the size of the palm of the hand over the left posterior thoracic region. There is great contraction of both visual fields.

REMARKS. This case we took to be one of hysteria with some element of neurasthenia. Malingering again seemed out of the question, for we believed it wholly improbable that an unsophisticated girl could simulate contracted visual fields, spinal tenderness, and segmental anæsthesia—stigmata of hysteria.

CASE IV.—L., man, aged thirty-seven years, enjoyed good health before the explosion. He was slightly burned about the face and greatly shocked, but at once began to assist the girls out of the building. He thought at first he would not be troubled in any way except by the slight burns. He complained that he was afterward troubled a great deal by nervousness, but he could not describe in anything like a definite manner in what this (nervousness) consisted. The chief burden of his complaint was concerning his eyes. He stated that since the accident he has been greatly troubled in reading, could read only a few minutes before becoming so fatigued that he is compelled to rest his eyes. The oculist who attended him testified at the trial that Mr. L. suffered from an error of refraction in each eye, and that aside from this

they presented nothing abnormal. He resumed work ten days after the accident, and has continued at it up to the present.

REMARKS. The symptoms of general nervousness and difficulty in reading were described by Mr. L. in such a vague and indefinite manner that we concluded they were largely or wholly simulated, and the oculist's declaration that errors of refraction existed in the eyes which could in no way have been caused by the accident only confirms this opinion. The very healthy appearance of Mr. L. at the time of our examination, and the fact that he has worked steadily since the explosion, also favored this view.

CASE V.—Man, aged forty-four years. He suffered severe burns about the scalp and face which were much swollen. These burns were much better at the end of three weeks. He did not work until three months after the accident. He tried, but failed. At times he has a good deal of headache. Sleep is restless, appetite poor; weight 123 pounds. He has a sense of being tired. He describes in a vague way certain confusion to which he is subject in the midst of his work.

REMARKS. There is no question as to the burns; the nervous symptoms were purely subjective, and while, possibly, they had some foundation in reality, they are probably a good deal exaggerated, and were not even, as described by the claimant, very severe.

CASE VI.—Girl, aged twenty-two years. She was in a highly emotional and excited condition immediately following the explosion. The next day her physician who called upon her found some elevation of temperature, and the following day some right ovarian tenderness. He thought there was inflammation in the right pelvis. She remained in bed three or four weeks. She worked for a short time, but in June, 1895, she was again taken down in bed with nervous dyspepsia and severe (crapulous) diarrhœa. Her physician attended her two weeks at this time. Her sleep has been greatly disturbed; she frequently awakens screaming. She suffers greatly from lack of energy and endurance, and has a tired, dragged-out feeling. Her mother (who was with her) states that the girl has changed greatly in disposition; that she is morose, depressed in spirits, and easily irritated by noises. The girl states that she frequently has most distressing dreams about the accident. She was evidently very nervous during our examination. She states that the right ovarian tenderness persists and troubles her a great deal. She worked from September, 1895, up to the present time, but in much distress.

REMARKS. There was in this case, apparently, some ovarian inflammation, and perhaps pelvic, too. Doubtless there was a severe shock to her nervous system, and considerable nervous irritability and prostration (even allowing for exaggeration) have resulted. The genuineness of the symptoms in this case can scarcely be doubted, for we should have to suppose too much if the theory of malingering were adopted. The symptoms suggest a condition representing an admixture of neurasthenia and hysteria.

Case VII.—A., girl, aged eighteen years. She received a severe burn on the left forearm, involving one-third of its circumference, and extending from the elbow to the wrist. She complained of no nervous symptoms.

Remarks. The negative observation that this girl, although having received a severe burn, suffered from no nervous symptoms is of interest, for it is quite likely that she received as severe a shaking-up as some of the others in the building and who afterward complained of nervous symptoms.

General Remarks on the Above-related Cases. Soon after the explosion damages were claimed in all these seven cases, and while some of the claimants suffered surgical injuries, the symptoms on which they rested their claims chiefly were those of a nervous character, except in Case VII., in which no nervous symptoms were complained of; and yet, curiously enough, this very claimant suffered the most severe surgical injury of any of these seven cases. These suits for damages were tried in March, 1896, more than a year after the explosion. During this interval the different claimants saw each other, visited the scene of the disaster, and talked over their injuries and the pending damage claims. Each claimant returned to work at the old shop after it had been repaired and as soon as he or she felt able. Mr. H. (Case I.), as has already been stated, assumed the position of watchman about the old shop three days after the accident, and continued in this position for two weeks, when the coma, which was followed by hemiplegia, supervened. Mr. H. also heard his physician testify at the trial that he could never get well, that his trouble was incurable, and that he would go from bad to worse.

Let us now briefly examine these psychical factors which were at work in these cases. In the first place, the explosion itself, no doubt, produced a very great psychical shock in each of these cases; and the association of the claimants with each other, their frequent visitations to the scene of disaster, their conversation with friends, attorneys, and physicians, and, lastly, the excitement incident to a lawsuit which they looked forward to for months; the hearing of more or less gloomy prognoses of their cases in court, constituted a most powerful series of psychical factors which were in the highest degree calculated to maintain and exaggerate neurasthenic and hysterical symptoms, or, indeed, set them up and retard progress toward recovery of individuals suffering from any affection. Surely, if there is anything at all in harmful mental suggestions, they are well illustrated in these cases. How far removed from ideal psychical conditions were those surrounding these unfortunate claimants!

In Cases IV. and V. we believed that there was very little basis of reality in the symptoms complained of, yet I would not like to say that

either of these claimants was a malingerer pure and simple. Each had received a tremendous shock and a surgical injury, and felt aggrieved at those responsible for them. Then the association and conversation with the other claimants probably led to a great deal of introspection or self-argument, the result being that each convinced himself that he had some nervous symptom much after the fashion that some persons argue themselves into certain feelings or opinions they wish to possess; *e. g.*, old fishermen and soldiers, etc., it is well known, often believe their own lies after they have told them a great many times. But in these two cases the ideas which gave rise to their symptoms were so feebly impressed on the consciousness that the symptoms themselves were vague, indefinite, and I had little doubt would disappear after the settlement of the damage claims.

The next case to be described was also a litigation case.

CASE VIII.—Man, aged fifty-four years, married, machinist. He was working at his trade, and in excellent health, when the accident about to be described occurred.

On April 1, 1895, he was struck by a "live" electric wire on the hypothenar eminence of the left hand, sustaining a shock which rendered him temporarily unconscious. When he recovered consciousness he felt numb and stiff all over his body. The next day he tried to work, but the day following he quit work on account of this feeling of being "numb and stiff." By the second day his hand had greatly swollen, and still more by the third day. A few days later the swelling subsided, leaving a small cicatrix. On the third day after the accident he experienced severe pains all over his body. His daughter states that after the accident she noted the metallic fastening of his suspenders had burned an imprint upon his back. Ever since the accident he has been daily subject to severe shooting pains along the spine and down the legs, and to a less degree in the arms. These pains often last several hours at a time. He states that he has lost his "snap" and energy, and that his power of endurance is greatly lessened; that his sleep is greatly disturbed, whereas formerly he always slept well. His wife states that since the accident he has become cranky and irritable, and is easily annoyed by trifles; that while it was formerly very easy to get along with him at home, this has, since the accident, become a very difficult matter, owing to this marked change in his disposition. She further states that he has lost all of his cheerfulness and is greatly discouraged; that whereas it has been his custom to indulge in sexual intercourse once a week, he has not attempted to have connection since the accident. His appetite is poor.

The first examination was made about three months after the accident. The man had a worried, distressed look, and, during the examination, broke into tears. His weight was 160 pounds (claims that before the accident he weighed 182 pounds). There was a tremulousness about the face and a tremor in his hands. Stripped, he exhibited no atrophy. There were no quantitative or qualitative electrical changes. Dynamometer: right, 60; left, 80. Both knee-jerks were feeble. Pin-pricks were not felt in the left leg or thigh, and tactile sense is somewhat

diminished over the member. He stated that there was numbness in this leg. There was no other sensory disturbance. There was marked tenderness on pressure over the spinous processes from the ninth thoracic downward. On the several occasions upon which I saw this man the burden of his complaint was always the same.

In the spring of 1896, one year after the accident, he accepted $900 in settlement of his claims for damages. I saw him in May, 1897, about thirteen months after this settlement, and noted no change in his appearance. He told me that the severe shooting pains, loss of endurance and sexual power, depression of spirits, and other subjective symptoms continued, and that he was still unable to resume work.

REMARKS. The only objective signs in this case were diminished knee-jerk, tremors, and the loss of pain-sense in the left leg (if this latter can be called an objective symptom). Yet the man's way of telling his story and his consistency in repeating it, his general appearance, and the corroboration part of his story received from his wife and daughter convinced me of the reality of his symptoms. Moreover, if the position were taken that the man was a malingerer, one would have to suppose that he was most skilfully acting a part, a thing I believed a man of his intelligence incapable of doing, even if he so desired. Finally, the symptoms from which this man complained are those we see as expressions of an exhausted and irritable nervous system, and I look upon his case as one of severe neurasthenia. The persistence of his symptoms thirteen months after the adjustment of his damage claims strongly bears out the views I have above expressed, and affords a strong argument against those who contend that cases like this, with scanty objective symptoms, are pure cases of simulation, and get well as soon as they have received damages.

The next case was the subject of a study by Dr. F. H. Edsall and myself,[1] on account of a most interesting condition of partial ophthalmoplegia which it presented, and which appeared to us to throw considerable light on the question of localization in the third nerve nucleus. For a description of these cases I quote from this publication.[2]

CASE IX.— H.T., aged fifty-two years; blacksmith; denies syphilis. He was in robust health and working regularly at the time he met with the accident about to be described, and to which he attributes all the symptoms from which he now suffers.

Four years ago he fell into a pit eight feet deep, sustaining a fracture of a rib and a number of bruises. He was not rendered unconscious by the fall, but states that he was very much "shaken up" by it, and was in a dazed condition for some time afterward.

The next day after the accident he made an effort to work, but found he was too stiff and sore to do so, and gave up the attempt. He states that he has from that day suffered from the symptoms from which he now complains, except that the pain from the broken rib and bruises soon disappeared.

[1] Annals of Ophthalmology and Otology, v., No. 2, April, 1896. [2] Op. cit.

Present condition. Short, stout, thick-set man. He walks with feet wide apart, taking short steps, carrying head well thrown back. (See the accompanying photograph.) The scleræ are seen between the upper lids and superior margins of the cornea. His speech is slow, measured, sometimes halting. (This symptom, too, appeared since the accident.) He complains of pain in the occipital region and along the entire spine. Muscular strength and power of endurance are greatly diminished. His finger movements are weak and clumsy, so that he finds it very difficult to button or unbutton his coat. The knee-jerks are normal. There are several points along the spine pressure over which causes much pain. There is bilateral superior rectus palsy

(almost complete), which accounts for the backward tilt of the head. While the internal recti act in a conjugate deviation of the eyes, they act little if at all in convergence. Marked (almost complete) palsy of the iris and ciliary muscles is present. The eye-grounds reveal no striking changes.

A recent examination, made in December, 1897, showed that the ophthalmoplegia had progressed. The inferior recti were almost totally paralyzed. Conjugate movement to the right and left was still preserved, but markedly weakened.

The peculiar gait, the stiffness and clumsiness of movement, and the occipital and spinal pains were still present, causing him great discomfort. The right leg and arm now showed marked spasticity and considerable loss of power.

Remarks. We have in this case progressive ophthalmoplegia and a most marked series of objective symptoms. To be sure, there is a possi-

bility that, after all, they may not have been brought about by the accident, and that their occurrence after it may have been merely a coincidence. Two points argue against this, viz.: (1) the statement of the man that very soon after the accident he began to hold his head tilted backward; and (2) the subsequent appearance of right-sided paresis and spasticity. All his other symptoms are subjective, and if the view be accepted that the eye symptoms resulted from the injury we have a most powerful argument for the genuineness of the subjective nervous symptoms. Then, too, it must be remembered that this was not a litigation case; and this fact, the man's age (forty-eight years at the time of the accident), and the apparently entire absence of a neuropathic temperament are further arguments in favor of this view. The foregoing considerations lead me to believe that both the eye and the general nervous symptoms resulted from the injury the man received almost five years ago. However the ocular palsies originated, whether through molecular death, or hemorrhages, or inflammation in the third nerve nuclei, *progressive degeneration of these nuclei has gone on since the accident.* Actual organic changes, then, being almost certainly at the bottom of the ocular palsies, and partial spastic hemiplegia having developed, it is altogether reasonable to suppose that the general nervous symptoms are to be accounted for by organic molecular changes in the finer nerve elements, and changes, demonstrable, too, by the aid of the microscope. In a case which presented very severe nervous symptoms following a traumatism, and in which the central nervous system was examined after death by Dr. F. X. Dercum,[1] no changes were noted. But the criticism was made by Collins[2] and others that the nerve-staining method of Nissl, had it been employed in this examination, might have shown changes.

From a clinical point of view, this case is difficult to classify. It is certainly not one of the hysteria, and to call it neurasthenia, even in part, seems like begging the question. It differs greatly, even aside from the eye symptoms, from any other case in the series. Widespread progressive degenerative changes in this man's central nervous system must be going on.

Case X.—A. C., aged twenty-one years, single, grocer; his father is rheumatic, and his mother has "nervous debility;" he worked regularly, and was always in good health except for occasional attacks of quinsy.

September 2, 1894, while sitting on a railing, he was, with a number of companions, precipitated backward fifteen feet to the street below, because of a sudden break of the rail. All saved themselves except the patient, who fell, striking a rock projecting from the wall, at the small of the back, alighting on hands and knees. A number of superficial bruises resulted. He arose at once to assist a companion who had sus-

[1] Transactions of American Neurological Association for 1895. [2] Op. cit.

tained a fracture of the knee-cap, and in whose welfare he became much concerned. During the two or three days following the accident he noticed nothing wrong. At the end of this time he began to stagger. This symptom increased steadily up to November, 1894.

The staggering was always worse after exertion, and almost disappeared after rest. Upon rising in the morning after a night's sleep he would not stagger at all. Toward evening (after exertion) the staggering would be very exaggerated, so that he was often unable to walk alone. Exactly at the time staggering began numbness appeared over the whole left side, including face, and increased, accompanied with motor palsy, on the same side. During this time he was eating heartily and sleeping very well, but says he went to closet seldom: occasionally a week would elapse between his visits. Often he could scarcely control bowels; when desire for stool came he was compelled to go very quickly. On several occasions he met with accidents, being unable to get to closet in time to save himself from disgrace. This bowel trouble lasted until the spring of 1895. In the middle of November, 1894, the numbness and loss of power on the left side were very marked, but with difficulty he could, after rest, walk a couple of squares. At this time he saw a physician, and while under treatment the numbness disappeared from the left side overnight, while asleep, only to appear in the right side, which was involved exactly as the left side had been, if anything, in a more severe degree when he awoke in the morning. This he attributed to the medicine he had been taking.

From this time he made slow and satisfactory progress until the spring of 1895, when the anæsthesia and palsy as well as the staggering had grown less—indeed, almost disappeared. For the next nine months his condition varied little. I saw him for the first time in January, 1896. At that time the chief burden of his complaint was the loss of power of endurance. He had a strong grip in either hand, but there was a certain clumsiness in the fingers of the right hand which prevented him from executing delicate movements with the same facility as formerly. In walking he gives the right leg a distinct swing. He states that he felt a distinct clumsiness in his leg, and believed that he was not nearly as strong in the right as in the left side. No contraction of visual fields; knee-jerks increased. When seen a month later he complained of stiffness and soreness in his leg, and said that the skin over his abdomen felt an inch thick.

On February 15th he started to run a laundry wagon, but gave up this occupation at the end of three days on account of a great increase of weakness in his legs (equal in both).

After March, 1896, he was lost sight of until January, 1897, when he reported that his condition was unchanged; that fearing his legs would give out he had not again attempted to go to work.

Remarks. There was no damage claim in this case, but if there had been, no doubt, since the symptoms were purely subjective, they would have been stoutly contested. So, in the absence of motive for simulation, it can be fairly assumed that this man is not a malingerer, for he has nothing to gain and much to lose by acting a part.

From a clinical point of view, the bizarre make-up of the symptom-complex, its shifting character, the hemianæsthesia with hemiplegia,

with the remarkable transference from one side overnight, are enough upon which to make a diagnosis of grand hysteria.

General Remarks on the Foregoing Cases. In considering now some lessons taught by these cases, one (VII.) may be eliminated from consideration, as it is not a nervous case at all, but has been introduced in this place for its negative value, viz., as an illustration of how an individual may receive severe physical injuries and escape entirely nervous symptoms through the same accident which will leave many complaining of severe nervous symptoms. Of the nine cases left for consideration, seven were litigation cases, two were not. Two of the seven cases I believe to be malingerers, or rather, instances of individuals who had by argument with themselves persuaded themselves of the presence of certain symptoms which had slight, if any, reality.

The remaining five of this litigation group I believed to be real sufferers, although the symptoms complained of were, for the most part, subjective. Exaggeration of symptoms, I believe, was made in most of these cases. It was made in Case X., which was not a litigation case, and I may add that it was so commonly seen in hysterical and neurasthenic patients with no "claims" that it ought, indeed, to make one hesitate in branding an individual as an impostor in whom it is observed. To demand that the objective symptoms alone be considered in examining these litigation cases is, to my mind, manifestly unfair; for certainly no one would maintain that the same rule be applied to the physicians examining ordinary patients. Most difficult, indeed, the problem is at times, but this short-cut way of disposing of it will not do in the face of the facts, well known to every physician, that subjective symptoms are often the most real, most severe, and most persistent of symptoms, and that frequently no objective symptoms, or only slight ones, accompany them.

To determine the value, then, of any set of subjective symptoms physicians should study most carefully *the manner in which they are related*, whether as a whole they fit into the description of any known disease or diseased condition. If they do so, the probabilities are very strongly in favor of their genuineness. Objective symptoms, even when they are few and minor in character, are of great importance in sustaining this view. Now, supposing that symptoms do present themselves in a group so that, ordinarily in a non-litigation case, we should say that they were hysterical or neurasthenic, or both, is it not plain that in the litigation case we should say the same of them? Those who demand that these subjective symptoms be thrown out, because they may be simulated, suppose the individual who presents them to possess consummate skill. For to me it seems that the feigning of a long train of neurasthenic or hysterical symptoms would show, on the part of the simulator, a fine knowledge of these symptoms and splendid powers of

acting—suppositions which could be entertained in only very exceptional cases. The successful simulation of nervous symptoms would require, beside this knowledge and skill, a degree of attention well-nigh impossible.

I have several times asked persons in good health who were strong believers in the "simulation theory," how they would go about it to illustrate in their own persons their contention that simulation was easy, and I have never found one who had more than the haziest of ideas on the matter.

It was then by taking into account the manner in which symptoms were described, the symptoms themselves, and the fact that, for the most part, they were those common enough to neurasthenia and hysteria, and the fact that an adequate cause for them existed, that I was led to believe that five out of these seven litigation cases were genuine sufferers such as they (in more or less exaggerated manner) described themselves to be. Two cases (IV. and V.) I believed to be malingerers because of the manner and vagueness of their descriptions of their symptoms. Cases IX. and X. are strong arguments in favor of the position I have taken in this paper. Had they been litigation cases, probably both would have been called malingerers by some of the astute examining surgeons, who, as matters are, would be content to grant them all the symptoms they claim.

The symptoms from which these two unfortunate men suffered drag along year after year, in Case IX. becoming worse, although it is in both cases clearly against the interest of the men to have them do so. In Case VIII., although his damage claims were settled over a year ago for $900, he continues to complain of the same symptoms which bothered him before the settlement. I regret that I am unable to state the effect settlement has had on the other cases.

From a clinical point of view these cases are, I believe, for the most part, properly designated neurasthenia or hysteria, since they present symptoms commonly seen in one or the other of these affections or both of them. Cases I. and X. appear to be excellent examples of major hysteria.

In certain of these cases (VI. and IX.), however, something more than, *or other than*, hysteria or neurasthenia exists. Certainly the progressive degeneration of the third-nerve nucleus and spastic hemiplegia are symptoms of neither affection. To explain this case and, perhaps, Cases VI. and VIII., one is tempted to adopt the views of Schmauss,[1] Leyden, Obersteiner, and Bikeles, who hold that death of nerve-substance may be produced by shocks without antecedent hemorrhage or inflammation. This theory would indeed better account for the general nervous symp-

[1] Centralblatt für Nervenheilkunde, August, 1894.

toms, aside from the progressive ophthalmoplegia seen in Case IX., than the hemorrhage or inflammation theories. The same would be true of Cases VI. and VIII. There are in this series far too few cases upon which to base any broad conclusions as to prognosis. But with two non-litigation cases (IX. and X.) going on several years without improvement, but, on the other hand, in Case IX. progressive deterioration taking place, and with another, Case VIII., complaining of all his old symptoms six months after the settlement of his damage claims, we have an ample refutation of the views that these nervous affections do not occur where there is no question as to damages and that prompt recovery always follows the settlement of damage claims. These three cases, however, I believe are to be regarded as unusually grave and offering exceptionally unfavorable prognoses. The same can be said of Case I. All the other cases at the time Dr. Shaw and I examined them stated that more or less improvement had occurred.

Conclusions. 1. Nervous symptoms complained of as following severe accident are, while often exaggerated, usually very real.

2. Simulation is rare and easily detected by the skilled neurologist.

3. To successfully simulate nervous symptoms is a difficult task, possible to only a few.

4. The symptoms in any case may be subjective wholly or chiefly.

5. The nervous symptoms set up are, as a rule, neurasthenic or hysterical, or both.

6. A certain number of cases suffer from symptoms not attributable to either of the above conditions.

7. An acutal degeneration of the nerve-substance is sometimes set up, and may progress.

8. While there is a strong tendency to recovery in many of these cases, the prognosis, in not a few cases, is grave, and in still others quite hopeless.

9. While the physical element in most of these cases is powerful, there are others in which it plays a very minor rôle.

10. Any name used to describe these nervous affections carrying the idea that they constitute a morbid entity is undesirable.

11. Some name, *e. g.*, "traumatic neuroses," if used to mean any nervous affection following a traumatism, would be useful.

A CONTRIBUTION TO THE STUDY OF THE MUSCULAR DYSTROPHIES.

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For a long time it was supposed that progressive muscular atrophy, as first systematically described by Aran in 1850, was a disease of uniform pathology, resulting from degeneration of the ganglion-cells in the anterior horns of the spinal cord. Later studies, however, have shown that such atrophy may result also in the absence of appreciable lesion of the spinal cord or of the motor nerves representing the peripheral continuation of the anterior ganglion-cells of the spinal gray matter. In explanation of the cases included in the second group it is assumed that the disease originates in the muscles themselves, constituting the so-called myopathies, in contradistinction to the cases of spinal origin, or myelopathies. In some cases, further, it had been observed that with motor weakness there was associated increase rather than diminution in the size of the muscles functionally concerned. To Erb,[1] in especial, belongs the credit of having pointed out that in both the cases with wasting and those with apparent hypertrophy the essential demonstrable lesion is the same, viz., increase in size of some muscular fibres, with diminution in size of others, and degenerative changes; and more or less increase in the interstitial connective and fatty tissues. Erb is, however, not entirely willing to admit that the changes in the muscles are purely myopathic, contending that they may depend upon impalpable nutritional alterations in the ganglion-cells of the anterior horns of the spinal cord not capable of demonstration with our present resources. He, therefore, proposes to designate this group of cases as muscular dystrophies, thus not committing one definitely to a theory of their pathology. Several types of the disease have been described, as, for instance, the idiopathic, the pseudo-hypertrophic, the juvenile or scapulo-humeral of Erb, the infantile of Duchenne, or the facio-scapulo-humeral of Landouzy-Déjérine, and the hereditary of Leyden; but it is doubtful if any useful purpose is subserved by this classification, inasmuch as the boundaries of the several so-called types are ill-defined, and many if not most cases present features of two or more varieties. It is probable that these several forms represent rather differences in degree and distribution than variations in essential character.

The muscular dystrophies are, in general, characterized especially by

[1] Neurologisches Centralblatt, October 1, 1883; Deutsches Archiv für klinische Medicin, xxiv., 1884, p. 467; Dystrophia muscularis progressiva, Leipzig, 1891.

their hereditary or familial distribution, their onset early in life, their preponderant occurrence in males, their progressiveness of course, the frequent presence of enlargement of some of the muscles affected, the disappearance of the mechanical irritability of the muscles, with loss of reflexes and quantitative changes in electric reactions. The disease usually sets in during the first years of life, and affects males rather than females. It is said to occur later in girls than in boys, to be in the former slighter in degree, milder in course, slower in progress, more protracted in duration, and less frequently fatal.[1] Sometimes an hereditary predisposition can be ascertained to exist, the transmission usually taking place through a healthy mother, while male children more commonly suffer. Often a familial distribution can be discovered, and occasionally the disease appears to be congenital. Finally, in a considerable number of cases the source of the disease entirely escapes scrutiny.

Weakness of voluntary muscles is commonly the first symptom noticed, and coincidently, or shortly afterward, the affected muscles are found to be wasted; although often, as has already been indicated, some of them are enlarged, and they are usually firm. In accordance with the distribution of the muscular changes there is impaired mobility of upper or lower extremities or of the face or of the trunk, or of several of these parts in more or less indifferent association. As the muscles degenerate, their reactions to mechanical, electric, and reflex stimuli diminish quantitatively; and late, at an advanced stage of the disease, the feebly opposed action of the less weakened muscles may result in contractures. The action of the sphincters remains unimpaired throughout. Sensibility is in no way deranged. The mental functions do not suffer as a part of the disease *per se*, and they are, as a rule, well performed.

The disease is of long duration and is progressive in course, though sometimes but slowly so. It is not in itself directly fatal, death usually resulting from some intercurrent or complicating or accidental disorder. Pulmonary complications are especially liable to result from weakness of the respiratory muscles. After death the nervous system is ordinarily found to be intact, only the changes spoken of as present in the muscles being found constantly.

The muscular dystrophies are to be distinguished especially from multiple neuritis and degeneration or inflammation of the anterior horns of the spinal cord. From all of these they differ in the distribution of the weakness and the wasting, the muscles of the hands and feet being rarely if ever involved in the dystrophies; in the absence of qualitative changes in the electric reactions of the affected muscles; in the firmness and sometimes the enlargement of the muscles; in the hered-

[1] Gowers: Diseases of the Nervous System, 1892, vol. i., 2d ed.

itary or familial distribution. From neuritis they differ besides in the absence of sensory disturbances. From chronic poliomyelitis or degeneration of the anterior horns of the spinal cord they differ further in the absence of fibrillary contractions; in their onset at an earlier period of life; in the absence of bulbar symptoms, as well as those of lateral sclerosis. Acute anterior poliomyelitis of infancy is a disease of rapid onset and of rather retrogressive than progressive course.

I have been able to collect from the preserved records of the Orthopedic Hospital and Infirmary for Nervous Diseases twenty cases of progressive muscular dystrophy, which, through the kindness of the physicians in charge, Drs. S. Weir Mitchell, Wharton Sinkler, and Morris J. Lewis, and of Dr. Thomas G. Morton, one of the surgeons, I am permitted to report in this communication.

CASE I.—M. E. B., a girl, aged three and one-half years, applied at the Infirmary for Nervous Diseases on October 23, 1873, when a provisional diagnosis of pseudo-hypertrophic paralysis was made. The notes state that the child was born at term, with weak limbs. Its appearance was puny, its abdomen prominent, and its chest convex anteriorly. Its left knee was contracted, and it was unable to stand without aid. The movements of the arms were preserved, though feeble. The left hand could be flexed only with difficulty. All of the movements of the legs were preserved. The posterior muscles of the legs were hard, and there was no wasting of particular muscles. The skin presented no mottling.

CASE II.—W. W., a boy, aged nine years, of German extraction, born in the United States, applied at the Infirmary on July 14, 1886, when the following history was obtained: There were three other children in the family, aged respectively fourteen, eleven, and two and a half years—all well. One child had died of croup. The father was alcoholic, and members of his family were tuberculous. He died suddenly seven months later. There was no knowledge of the existence in any branch of the family of any disorder like that from which the patient was suffering, nor was there other history of nervous or muscular disease. In addition to the usual diseases of childhood the boy had had an attack of pneumonia at the age of four years. At the age of six years it was noticed that the child fell frequently, but no significance was attached to this, as he appeared well and strong. From this period there had been increasing difficulty in walking, without any other manifestation. The boy was fairly well grown for his years. His head appeared small, and the forehead was narrow. The expression was intelligent and the general appearance healthy. The chest was suggestive of rickets; the costal cartilages were slightly prominent; the back was bowed; the abdomen was protuberant. The spine appeared to be slightly curved toward the right. The dorsal muscles were prominent. The calves were large, while the thighs, in comparison, looked small. In rising from the dorsal decubitus the mode of action was quite characteristic. Fourteen months after these observations, it was noted that in walking the boy leaned backward and swayed from his hips. The spine was greatly curved forward in the dorsal and lumbar regions. In arising from the stooping posture the boy climbed up

his thighs. Great overgrowth of the calf-muscles was noted, and these were hard. The overgrowth of the thighs was not marked, while the chest and arms were thin. The knee-jerks could not be elicited, nor could the muscle-jerks in the thighs. The tendo-Achillis jerk was active, the more so upon the left, and the cremasteric reflex was present; so also was the front-tap contraction. A slight epigastric reflex could be elicited. The elbow-jerks were present, though feeble; the muscle-jerks in the arms were active. The chin-reflex was marked. Both testicles were retracted.

Case III.—A. H., a boy, aged eleven years, entered the Infirmary January 9, 1888. His parents were alive and healthy. They had borne eight children, all of whom were living. The patient had been in good health until the age of eight years, when it was noticed that in running he often tripped and fell. Later, he would fall even in walking. Finally, at the age of ten years, he lost entirely the power of walking. At the same time his knees began to grow stiff. The boy was quite unable to stand, but he could sit up when placed in a chair. The knees were flexed and stiff, the feet inverted. The legs could be moved but slightly. The boy was large, weighing eighty-two pounds. The muscles of the calves and thighs seemed most enlarged, the arms next in degree, and the face, abdomen, and chest least. The gluteal creases were almost completely obliterated. The masseter muscles were hypertrophied. The spinal column presented an antero-posterior curvature. The knee-jerks were wanting on both sides. The mental condition was poor.

Case IV.—H. D., a boy, aged eight years, presented himself at the Infirmary on October 22, 1888, on account of undue readiness of fatigue in walking. The gait, however, presented no obvious abnormality, and station was steady. There was no complaint of pain. When seated on the floor, the boy was unable to arise without catching hold of something, or by climbing up his thighs. In ascending stairs he placed one hand on the step above him and the other on his knee. It was stated that he had been stout, although he had of late grown thinner. The calves of the legs had not increased in size. Their muscles responded normally to faradic stimulation. The knee-jerks could not be elicited, even with reinforcement. The plantar reflexes also were absent, while the cremasteric and the abdominal were preserved. The angles of the scapula were prominent. A line dropped from the spine of the seventh cervical vertebra fell behind the coccyx, although within the plane of the buttocks. The boy's father had died of pulmonary tuberculosis. A brother and two sisters were living, and, together with the mother, were healthy. No other member of the family had suffered from a disorder similar to the patient's. The boy had had scarlet fever at the age of four years. He had never walked like other children, always presenting a strut. As he grew older he manifested a tendency to fall on attempting to run. When seen a year after his first visit to the dispensary it was learned that for a period of five months the calves had been noticed to be increasing in size, and they were found to be large and hard. At this time, also, the right supraspinatus muscle appeared a little larger than the left. The boy walked well, although he carried his shoulders far back.

Case V.—F. M., a boy, aged eleven years, applied at the Infirmary for Nervous Diseases on December 5, 1888, walking with his legs spread

far apart, bending the knees but little, and going slightly on the tips of the toes. The station was good, although the patient complained of giddiness when the eyes were closed. After being seated upon the floor he arose by climbing up his thighs in characteristic fashion. He presented decided wasting of the thigh-muscles, particularly anteriorly, while the calves were markedly enlarged. To the touch the latter seemed hardened and smooth. The rhomboids were wasted and the supinator longus on the right was to the eye smaller than that on the left. The knee-jerks were absent, as were also the muscle-jerks in the thighs. There was an older and a younger child in the family; both were well. The sister, who was married, had two healthy children. The patient had had scarlet fever at the age of five years, being kept abed five weeks. Six months later the legs were noticed to be weak, though they were never observed to be unusually large.

Case VI.—L. S., a boy, aged ten years, applied at the Infirmary on May 3, 1889, when it was related that he had walked naturally up to the age of two years, when he suffered from measles and pneumonia. Immediately on recovering he was found to be weak in the legs. He did not walk at all for four months, and thereafter only imperfectly. The muscles wasted gradually over the whole body. At the time of coming under observation the calves were said to be not as large as they had been. They were hard, and larger in proportion to the other limbs than would be expected from the general wasting. It was said that the boy fell in the street, but he was able to arise from the floor without aid. The knee-jerks were wanting, and no muscle-jerks could be elicited in the thighs. The ankle-jerks were present, though feeble. Faradic irritability was lessened in the extensor muscles of legs and thighs. With the galvanic current K. Cl. C. = An. Cl. C. in the extensors of the legs.

On December 4, 1890, the patient was unable to stand without support. It was thought also that the arms were now weaker than they had been. There was no pain and no tenderness over nerve-trunks. No derangement of tactile or painful sensibility could be detected. There was no local palsy, and co-ordination in the upper extremities was good. The cremasteric and abdominal reflexes were active. The thighs appeared to be turned inward, but there were no contractions.

Case VII.—J. P., a young man, aged twenty-two years, applied at the Infirmary on September 11, 1889. As a school-boy he had noticed, independently of any preceding illness, that running was difficult and would be attended with a fall. For as long a time as the patient could remember, his thighs and upper arms had been thinner than the rest of his body. When he presented himself he appeared to be a fat, well-built man. He manifested considerable difficulty in rising from his chair, putting his hands on his knees to assist himself in getting up. He walked slowly and would fall on running. When down he had much difficulty in rising. He was unable to extend the legs upon the thighs, while some power of flexion was preserved. There was also loss of power in the upper extremities. With the hands the man was unable to resist a weight of three pounds. The legs and arms were small as compared with the trunk. The calves were large as compared with the thighs. In sitting down he dropped into his chair, and he arose in a characteristic manner. To the sense of touch there was no obvious difference between the superficial and the deep tissues. The knee-jerks were

wanting. There was no pain, and sensibility was preserved. Tremor was developed on extending the hands. The patient was employed as a bag-folder, and his general health had always been good. He was not addicted to the use of alcohol or to sexual excesses, but he chewed a good deal of tobacco. His mother and two sisters were dead.

Case VIII.—J. B., a boy, aged seven years, applied at the Infirmary on June 22, 1892, presenting a waddling gait, beginning talipes equinus, increasing hollowness of the back, and large, hard calves. The knee-jerks could not be elicited, and he arose from the floor in a characteristic manner. It was learned, further, that the child had been delivered instrumentally at term, and had been principally bottle-fed. He began to walk at the age of two years, and made slow progress, falling often in his attempts. His father was living and had suffered from chronic dysentery for nine years. The mother was living, in good health. He had also a sister and two brothers, all in good health. There had been no death in the family.

Case IX.—E. W., a girl, aged nine years, was referred to the Infirmary by Dr. G. S. Thornton, of Lewisburg, Pa., on October 19, 1892. Her father had died of heart disease. Her mother and five other children in the family were all healthy. A paternal uncle had been insane. The child was born without difficulty and without instrumental aid. She suffered no injury and had never had a convulsion. She began to talk at an early age, but was mentally not as far advanced as other girls of her age. Her general health was good. She had broken her right hip when six years old. The child was brought on account of weakness of the left leg, which the mother thought dated from birth. She never crawled, and did not walk until the age of three years, when the trouble with the left leg became especially noticeable. The child fell frequently, but wasting was never observed, and there was no weakness in the arms. In walking it was noticed that the child favored the right leg, while in standing the pelvis was tilted toward the left. In picking up objects from the floor she supported herself with the hand on the left thigh above the knee. She was unable to arise from the floor without the aid of a chair or other support—straightening the leg first, then lifting herself with the aid of a chair, and finally gaining the erect posture by supporting herself on the left thigh with her hand. There was a tendency to turn the feet inward. The thighs were wasted and flabby, but the calves were hard and large. The spine was very hollow—S-like. Station was steady. The knee-jerks could not be elicited, even with reinforcement. The tendo-Achillis jerk was present. Elbow-jerks and chin-jerk were wanting. The muscles of the thighs and legs displayed marked quantitative changes in response to electric stimulation, but no qualitative alteration.

Case X.—C. S., a boy, aged nine years, was referred by Drs. E. S. Riggs and G. A. Mueller, of Allegheny, Pa., to the Infirmary on March 10, 1893. The child's parents were both dead from nephritis. Four sisters were living and well. There was no knowledge of the existence of nervous disease in the family. The boy had had measles in childhood, but no other acute disease. It was thought that he had never been as strong as other children. He was able to stand alone when a year old, and walked soon afterward. He had several falls from chairs, etc., but seemed to suffer no harm therefrom. When he came under observation it was related that he had been gradually losing power in

hands, arms, and legs. For seven months he had been unable to walk up stairs without assistance. The boy looked healthy, and his muscles seemed to be rather over-developed. His appearance was bright and his intelligence and disposition good. All of the bodily functions were apparently well performed. The sphincters were under perfect control. The muscles were large and solid, and movement at all of the joints was free. The muscles of the calves, however, were contracted, so that the feet were thrown into a position of talipes equinus. In standing, the body was supported upon the toes. Walking was impossible on account of the extreme equinus. The spine was curved in lordosis, and the muscles of the back appeared hypertrophied. All of the muscles responded normally to electric stimulation. Sensibility was preserved. The knee-jerks could not be elicited. The skin-reflexes were normal. The pupils were normal. The treatment consisted essentially in massage, and as soon as the physical condition of the child was favorable the tendo Achillis of each leg was divided. The feet were straightened as a result of the operation, but the boy was unable to stand unsupported, apparently from want of power in the muscles of the back and thighs. Later, a plaster-jacket was applied, but rendered little service. The reaction of the muscles to faradism gradually diminished.

CASE XI.[1]—P. C., a boy, aged eleven years, was admitted to the Infirmary on March 17, 1894. It was learned that his father was dead —from what cause could not be ascertained—while his mother was living and in good health. A cousin and an aunt had died of pulmonary tuberculosis. At the age of two years the boy suffered a severe illness which was designated a "cold on the chest." This was followed by a cough, for which cod-liver oil was prescribed. Muscular weakness was first noticed at the age of seven years. Upon admission to the hospital the boy was thin and poorly nourished. He walked with a peculiar gait, the belly being thrown forward and the body rolling from side to side as if to balance some instability of equilibrium. He was compelled to support himself with the nearest object at hand. The knee-jerks could not be elicited, and no anæsthesia could be discovered. The calves of the legs were hard and tense. On attempting to arise from recumbency in the dorsal posture, the boy would roll upon one side, then push himself backward upon his feet with his hands, which he used to support himself with, and continue to rise by resting on his knees and climbing up his thighs. The gait was waddling, the abdomen protuberant, the shoulders thrown back. The calves were very hard, the feet reddish and purple. The boy could stand on his toes, though not on his heels, and he could not jump. The thighs were moderately soft. There was no bellying of the biceps on flexing the arms. The arm-muscles were generally wasted, those of the upper arm more so than those of the forearm. The pectorals were wasted and the latissimi dorsi markedly so. The supraspinati and infraspinati were hard. The scapulæ were markedly alar. The neck-muscles were well developed, though wasted. There was no wasting appreciable in the face and none in the tongue. The knee-jerks could not be elicited, even with reinforcement. Elbow-jerks and triceps-jerks

[1] This case and Case XII. are depicted on page 865 of "A Text-book on Nervous Diseases," edited by Francis X. Dercum, M.D. (Fig. 284).

were enfeebled. The abdominal reflex was present, the plantar reflex wanting. There was no relaxation of the ankle-joints. The chin-jerk was present. Muscle-jerks could be elicited nowhere. The spine was straight laterally, but there existed lumbar lordosis which could be obliterated by extension. In going down stairs the boy clung to the wall or baluster, and further supported himself by placing one hand on the knee. The calves remained hard. The glutei were normal. The infraspinati were small, but hard. The triceps, biceps, and deltoid appeared normal. It could not be determined whether or not the costal portions of the greater pectoral muscles were normal. The tongue and face were normal, but much fuller than they had been, as a result of the general nutritive improvement. The muscles on the posterior aspect of the left thigh were harder than those on the right. Lifting the patient with the hands in the axillæ brought the shoulders up to the ears. The lordosis, which was marked, was obliterated by the sitting posture, which developed a slight kyphosis. In walking, the gait was rolling and acquired more of the tip-toe character than it had formerly possessed. Fibrillary muscular contractions could not be detected. Cerebral activity was good. There was slight mottling of the legs. Factitious urticaria was readily induced. No electric change was noted.

Case XII.—A. C., aged nine years, and a brother of P. C., was also admitted to the Infirmary on March 17, 1894. His symptoms, which were much the same as in the case of his brother, though less pronounced, had also been present from the age of seven years, when he began to waste and to become stiff on attempting to walk. The gait was waddling, the belly protuberant. The spine was straight laterally, but presented lumbar lordosis, which could be obliterated by extension. The knee-jerks were absent; also the plantar reflex, the biceps-jerk, and all muscle-jerks. The abdominal reflex and the chin-jerk were present. The calves were soft, but somewhat enlarged. The thighs felt mushy and the arms were generally wasted. The latissimi dorsi were wasted. The supraspinati and infraspinati stood out well. The neck-muscles were normal. There was no appreciable wasting of the face or tongue. The movements of the legs and arms were all present, but enfeebled. In arising from recumbency in the dorsal posture the boy climbed characteristically up his thighs. He could still do many things his brother could not do, his condition being not so far advanced. The glutei were firmer and more prominent than usual. The infraspinati were prominent and hard. The triceps, biceps, and deltoid were normal. No change was demonstrable in the pectoralis. The calves were not so firm as his brother's. The scapulæ were somewhat alar. The boy could be lifted by the shoulders much more readily than his brother. The erector spinæ muscles appeared enlarged. The lordosis was more marked and the abdomen more protuberant than in the elder brother. The patient was able to jump from the floor and also to stand on his toes. He was able, when requested, to arise from the floor without supporting himself with his hands on his legs, but he always grasped the thighs if left to himself. The knee-jerks, though absent spontaneously, could be brought out by reinforcement. Electric examination disclosed enfeebled response of the peronei to faradic stimulation, without galvanic alteration.

Case XIII.—P. T., a girl, aged seven and a half years, was referred to the Infirmary by Drs. Peeples and J. B. Walker on April 6, 1894.

Her father and mother were well, and there had been no miscarriage. Her grandfather had been paralyzed at the age of eighty years. Her only brother had died of cholera infantum. The patient was the first child, and was born at term after an easy labor, being delivered without instrumental aid. She appeared to be mentally up to the normal standard, but was always weak physically, though there was never any absolute paralysis. The child was nursed at the breast, and had never had a convulsion. It began to speak when a year old, and articulation had always been distinct. At the age of one year it could sit alone readily, but was unable to creep. All movements seemed weak, but the members were never rigid. At the age of a year and a half the child began to "paddle" when seated, the arms being too weak to aid in progression. It moved about thus by sliding until the age of three and a half years, when it laboriously began to creep about on hands and knees. Neither calves nor other part of the body ever seemed enlarged, nor was wasting ever noticed. Power was gained gradually. The child began to walk at the age of three and a half years, and kept on steadily improving, especially after treatment with electricity was instituted at the age of four and a half years. The girl appeared intelligent, spoke well, and displayed a good vocabulary for her years. Station was steady. She walked with her toes turned in and with more or less dragging of her feet. She was unable to go up stairs unaided on account of weakness. With considerable difficulty she could extend her arms high up over her head. She could not arise from the floor without aid—*e. g.*, as from a chair, against which she sometimes pressed her head to gain additional support. Without further assistance she could only arise to her hands and feet. The grasp of the hands was poor. The muscles of the arms were flabby. The elbow-jerks and the muscle-jerks were wanting. The knee-jerks were absent. There was lumbar lordosis. The thighs were proportionately larger and harder than normal. The response to faradic stimulation was lessened in the extensors of the forearms. The head was well shaped. The pupils reacted to light and in accommodation. Hearing was good. The sphincters were under control. All other organs seemed normal.

Case XIV.—C. C., a boy, aged eight years, was brought to the Infirmary on April 13, 1894. The parents were healthy. Of three other children in the family one was dead from whooping-cough, one from pneumonia, and one was healthy. There had been one miscarriage. The patient was born at term, after a hard labor, the feet presenting; but no instrument was used. He was vigorous at birth, weighing ten pounds. He was nursed at the breast until he was eighteen months old. He began to talk at the age of twenty months, and to walk at the age of twenty-one months. He had one convulsion in early infancy, and an attack of mumps when two years old. At the age of three or four years the mother noticed that the child had difficulty in getting about, but she had never observed whether the muscles were unusual in size or consistency. The boy was brought on account of difficulty in walking and in arising, with obvious weakness. In ascending stairs he was compelled to support himself with his hands upon his thighs. The gait was somewhat waddling, and on arising from the floor the child climbed up his thighs. The calves were increased in size and unduly firm. The thighs and arms were weak and their muscles wasted. The supraspinati and infraspinati were hard. The movements of the arms

and legs were preserved, but enfeebled. The ankles were relaxed. The knee-jerks were absent and could not be brought out by reinforcement. The elbow-jerks were absent. The chin-jerk was present and reinforcible. The plantar reflex was active. The cremasteric reflex and the abdominal reflex were present. None of the muscle-jerks could be elicited. The grasp of the hands was exceedingly weak. The patient could not jump from the ground. There was no wasting of the face or tongue. Sensibility was preserved. The spine presented no lateral deflection, although there was slight lumbar lordosis. The sphincters were perfectly under control. Faradic irritability was lost in the peroneal muscles. The teeth were serrated, but not notched. The foreskin was elongated, but not adherent. Urine was passed frequently, and at night into bed. The ribs were free from beading.

Case XV.[1]—J. R., a boy, aged ten and a half years, presented himself at the Infirmary on January 4, 1895. He had been born at term without complication, and during infancy had been nursed at the breast. At the age of nine months he had whooping-cough and shortly afterward chicken-pox. He learned to speak at the usual time, and his teeth appeared normally, without serious difficulty. No abnormality was noted until the child should have walked, but failed to do so. For a time, at about the age of two years, he was able to crawl and creep, but he had never walked unsupported. He had tried crutches and braces, but without real assistance. For five years he had been using a tricycle. He was unable to set himself in motion, but once started he could continue to propel himself. His intelligence was good. He had never had a convulsive seizure. The motor inability had grown progressively, and the child was utterly unable to stand without support. When seated, the lumbar spine projected forward, leaving the sacrum prominent. The legs and feet could be moved feebly, and the thighs also, though in less degree. Flexion of the thighs was particularly weak. The legs and feet were intensely cyanotic; in some places bluish, in others pinkish, in still others deep red. They were also cold, and their cutaneous covering rough. The calves of the legs and the buttocks were distinctly full and firm, but not indurated. The musculature of the rest of the body was spare. The child was unable to flex the trunk forward, and rotated the spine little and with difficulty. The movements of the upper extremities were well executed. The knee-jerks could not be elicited; nor could the muscle-jerks in the lower extremities. There was no contralateral contraction of the thighs; no ankle-clonus; no tendo-Achillis reflex. The epigastric, abdominal, and cremasteric reflexes were active; the gluteal less so. The erector spinæ reflex was good. The elbow-jerks were preserved, though feeble. The chin-jerk and the infra-orbital reflex were present. The pupils reacted normally. Tactile, painful, and thermal sensibility was everywhere preserved, though more acute in the upper than in the lower extremities. The mechanical irritability of the muscles of the trunk and upper extremities was increased; in the lower extremities it was wanting. The reaction of the muscles to faradic stimulation was enfeebled, especially in the lower extremities. The head appeared rather large; the ears

[1] This patient was exhibited to the Philadelphia Neurological Society on February 25, 1895, and a detailed account of his case appeared in the Journal of Nervous and Mental Disease, May, 1895. I have learned of his death since, but am uninformed as to its cause. No autopsy was held.

were prominent. The sphincters were under control. The grasp of the hands was exceedingly feeble, but seemed to be alike upon both sides, although the parents believed the right side to be the weaker. The movements of the upper extremities were free from ataxia. The disposition was bright and cheerful. There was no ocular abnormality. Some of the tendons of the feet had been operated on, but a tendency to valgus remained. The boy had three brothers, who were well and free from obvious abnormality. One brother had died soon after birth in consequence of injuries received during parturition as a result of dystocia due to unusual size. One sister had died at the age of four years, having had spinal curvature and finally meningitis. Another sister died suddenly at the age of nine months during dentition.

Case XVI.—W. G. C., a boy, aged eight and three-quarters years, was referred by Dr. Ellis, of Iron Hill, Md., to the Infirmary on March 15, 1895. The child, without morbid hereditary predisposition, was born at term after a difficult labor, not, however, requiring the aid of obstetric instruments, and, so far as known, without injury to the head or other part of the body. He was nursed at the breast and escaped the usual diseases of childhood. At the age of one year he fell from his coach, but suffered no appreciable injury. He began to walk when seventeen months old and to talk soon afterward. The family noticed that the boy, in attempting to walk, failed to do as well as other children of the same age; and also that he did not use a spoon well. He seemed well-developed, and the family noticed only the progressive weakness. At the age of two and a half years the boy had some difficulty in micturition, but this was corrected by the administration of belladonna. At one time he was unable to ascend steps on his feet, without placing one hand on the knee and with the other catching hold of some support. When he came under observation he was able to go up stairs only on his hands and knees. In fact, he could arise from the floor only with aid. At one time he arose by climbing up his thighs. In walking he held his legs far apart, apparently as if in an effort to maintain his equilibrium. The station was quite good, even with the eyes closed. In standing, the spine presented a marked anterior curvature in the lumbar region. There was no lateral deviation. The muscles appeared to be fairly developed, but were generally flabby, with the exception of the calf-muscles, which were large and hard. The latter measured eleven and one-quarter inches in circumference on the right and eleven and five-eighths inches on the left. The arm-muscles were not unduly large for a boy of his age. He was able to move his extremities in all directions, but without vigor. There was slight foot-drop. The grasp of the hands was exceedingly feeble. Sensation was normal. The knee-jerks were wanting. The plantar reflexes were present, as were also the tendo-Achillis reflexes; the cremasteric reflex was present on the left, wanting on the right. The abdominal and thoracic reflexes were wanting. There was no humping of the muscles of chest, arms, or thighs when struck a smart blow. The pupils were reactive to light. The fundus presented no lesion, and there was no weakness of any of the internal or external ocular muscles. The feet were cyanotic, but not mottled.

Case XVII.—N. M. J., a girl, aged sixteen years, of Irish parentage, born in Pennsylvania, presented herself at the Infirmary on September 4, 1895. Her mother had died at the age of thirty-nine years

from " inflammation of the bowels." Her father was said to be " very delicate " and suffered from cough. There were four other children in the family, aged twenty-two, twenty, fourteen, and twelve years, respectively—all in good health. There was no family history of nervous diseases, and all of the relatives were free from any disorder like the patient's. The girl herself had never been seriously ill, except for an attack of measles at the age of two years, one of whooping-cough at the age of four years, and one of influenza at the age of twelve years. Menstruation had not yet appeared. On examination the latissimi dorsi muscles were found wasted, with projection of the angles of the scapulæ, while the supraspinati and infraspinati appeared to be overdeveloped. The movements of the right arm dependent upon the deltoid were somewhat restricted. The calves of the legs were enlarged and hard. The patient was unable to arise from the recumbent posture even by climbing up her thighs. At times she would stumble and fall and be unable to get up. The first symptom had been noticed four years previously, and consisted in a flapping forward of the feet in walking, as though they were unduly heavy. Station was steady. The thighs were poorly developed and flabby. The knee-jerks were absent and could not be brought out by reinforcement. The pupils were equal and reacted to light and in accommodation. The scapulæ were very small, and presented an alar appearance. The arms could not be raised above the level of the shoulders unless carried forward. Movement at this joint was attended with a sense of crepitus, but no pain. The muscles of the back were greatly wasted. The buttocks were firm. Following the attack of influenza the patient was never quite strong. Shortly afterward she was noticed to rock from side to side in walking. Then the left scapula was observed to protrude. Next the arms became weak, the right more pronouncedly so. Finally, the wasting became general. The girl had previously been round and plump. While under observation it was noticed that the movements of the left side of the face were imperfect. The girl had never been able to whistle. The tongue was protruded median. In closing the eyes slowly the left lagged behind. The electric reactions presented no qualitative change.

Case XVIII.—J. W., an unmarried bass-drum maker, aged twenty-four years, born in Pennsylvania, applied at the Infirmary on October 16, 1895. It was learned that during childhood he had fallen from a second-story window, striking his head. His memory was poor after this, but there was no convulsion or other consecutive disturbance. The patient had had the diseases of childhood, and also an attack of variola at the age of ten years. He denied all history of alcoholism or venereal disease. His father had died of pulmonary tuberculosis. He presented himself on account of general muscular weakness, which had been present since childhood, when he experienced difficulty in getting up from the recumbent posture. On examination the calves were found to be large and hard. The left leg appeared to be the weaker. In walking, the abdomen was thrust forward, most of the weight of the body being brought upon the heels. In arising from the recumbent posture the left hand was rested upon the left thigh. To unaided observation there was no difference between the two sides of the body. The supraspinati and infraspinati were apparently increased in size. The spinal column was free from abnormal curvature. The knee-jerks could not be elicited. The elbow-jerks were preserved. The pupils

reacted well to stimulation. The chest protruded in pigeon-fashion. Heart and lungs were normal. Co-ordination was good and station was fairly steady.

CASE XIX.—E. J. R., a male, aged nineteen years, was admitted to the Infirmary on October 21, 1896. It was learned that his father had died of pneumonia after an illness of one week, while his mother had died of "consumption of the stomach." A brother and two sisters were living and in good health. Two sisters had died of some cerebral disorder. A maternal grand-uncle was thought to have had the same disease as the patient, which was finally transformed into elephantiasis, for which amputation was performed. The father of this relative was supposed to have passed through a like series of disorders. The patient had erysipelas at the age of five years. At about the same time he fell a distance of twenty feet, landing upon his back. He did not lose consciousness and was all right in a short time. He struck the top of his head, without, however, suffering a laceration. At the age of five years weakness of the legs was noticed in ascending stairs, when the hands would be placed upon the knees for support. For three years the patient had remained indoors on account of his disability. In walking he stood upon his toes, without bringing the heels down. He could not rise from the floor without support. At an earlier period of his disease he was accustomed to raise himself by crawling on his knees. Most of the muscles were very much wasted. The calf-muscles, however, were quite full and firm, and the feet in a position of slight equino-varus. Neither knee-jerk nor ankle-clonus could be elicited. The tendo-Achillis reaction was active on both sides. Feeble muscle-reactions could be elicited in the upper extremities. The plantar reflexes were wanting on both sides. When the plantar aspect of the arch of the foot was irritated the external thigh-muscles contracted (vastus externus), in greater degree on the left. The cutaneous reflex of the great toe could not be elicited. The cremasteric reflex and the epigastric reflex were wanting, while the abdominal reflex was preserved. Sensibility was preserved. The face appeared to be uninvolved and the tongue was of ordinary size. Speech was not affected, and there had never been a convulsion. The action of the heart was rhythmic, and its sounds were clear. Seated in bed, the patient was able to raise his arms above his head, when the atrophy of the pectoralis major, and especially its lower portion, was made conspicuous. The scapulæ exhibited a little displacement, but assumed the alar attitude when the arms were raised. The sphincters were under perfect control.

CASE XX.—G. K., a watchmaker's apprentice, aged twenty-two years, applied at the Infirmary on January 4, 1897. He related that his father was dead in the sequence of an apoplectic attack. His mother was alive and well. He had two sisters, who also were in good health. Neither he nor his mother had knowledge of disease similar to the patient's or of other nervous diseases in the family. The patient had been born at term, without complication, after a pregnancy attended with anxiety on the part of the mother. He had suffered from chicken-pox and mumps in early childhood and from measles at the age of twelve years. Dentition set in early and was uncomplicated. The child learned to speak early and began to walk when a year and a half old. It was noticed that he fell often between the age of three and five years. Several of the falls left cicatrices upon the forehead. There

had never been a convulsion. The patient used no tobacco and indulged only moderately in alcohol. Between the age of eight and thirteen years he had been an inmate of Girard College. The first abnormal manifestations were noticed after the attack of measles at the age of twelve years, and consisted in difficulty in ascending stairs. To accomplish this the patient was compelled to support himself with his hands on his knees and thighs. At about this time, or shortly afterward, difficulty was experienced in arising from the ground. These troubles grew gradually worse, although they had been practically stationary for four years. During this period the patient had walked on his toes. He found difficulty in the use of a lathe which necessitated the use of his feet, and also in cutting metals with his hands. The knee-jerks were absent. The tendo Achillis was shortened on both sides, but its reflex contractility was preserved. The plantar reflex was wanting, while the cutaneous reflex of the great toe was present, and irritation of the sole of the foot was attended with contraction of the flexor muscles of the thigh, more vigorously upon the left than upon the right. The muscle-jerks generally were wanting in the lower extremities. The thighs were greatly wasted, while the calves were greatly enlarged in comparison, and were firm. The buttocks were wasted, as were also the latissimi dorsi and the pectorals and all of the muscles of the upper extremities generally. The cremasteric, abdominal, epigastric, and erector spinæ reflexes were maintained. Feeble muscular and tendinous jerks could be elicited in the upper extremities. The chin-jerk was preserved. The usual lumbar lordosis existed. Sensibility appeared to be preserved. The action of the heart was rhythmic and its sounds clear. The complexion was sallow. The tongue was coated and of ordinary size. The action of the sphincters was controlled. Sexual desire was preserved, and occasional seminal emissions occurred. The appetite was moderate, the bowels fairly regular. Sleep was good; the memory was preserved; eyesight was good with the aid of glasses. Hearing also was good. The electric examination disclosed only quantitative changes.

It will be noticed that the cases are of mixed type, with a predominant tendency to muscular pseudo-hypertrophy. Of the whole number, but four occurred in females—20 per cent., and a proportion of 1 to 4 in males. Gowers,[1] in speaking of pseudo-hypertrophic paralysis, gives the proportion as 1 to from 4 to 7; idiopathic muscular atrophy, he states, is less uncommon in females. In 24 cases of pseudo-hypertrophy seen personally[2] he found the proportion 1 to 7 (3 females, 21 males), and among 220 cases collected from various sources the proportion was 1 to 6.3 (30 females, 190 males). Among 84 cases Poole[3] found 11 in females and 73 in males (1 to 6.6). Eichhorst[4] quotes Seydel as having found among 125 cases 22 in girls and 103 in boys (1 to 4.7).

So far as could be ascertained, the first symptoms in the cases here

[1] Diseases of the Nervous System, 1892, vol. i., second edition.
[2] Pseudo-hypertrophic Muscular Paralysis. A Clinical Lecture. London, 1879.
[3] New York Medical Journal, 1875, xxi. p. 569.
[4] Handbuch der speciellen Pathologie und Therapie, Bd. iii., 4. Aufl., 1891.

reported were noticed in none later than the thirteenth year; in 2 at birth; in 2 during the first year; in 2 during the second year; in 2 during the third year; in 2 during the fourth year; in 3 during the sixth year; in 1 during the seventh year; in 3 during the eighth year; in 2 during the ninth year, and in 1 during the tenth year. The ages of the patients at the times when they came under observation were as follows: three years, 1; seven years, 2; eight years, 3; nine years, 4; ten years, 2; eleven years, 3; sixteen years, 1; nineteen years, 1; twenty-two years, 2; twenty-four years, 1. In only two of the cases (XI., XII., in brothers) was it definitely ascertainable that similar disease existed in any other member of the same family, although in one other case (XIX.) the statement, which, however, could not be verified, was made that a great-grandfather and a grandfather had suffered from a like disorder. These findings are not in accord with the results of the analysis made by Gowers,[1] of 220 cases, of which 102 were isolated and 118 occurred in 39 families. Erb[2] gives the proportion of hereditary and familial cases as 56 per cent. In six of Poole's cases[3] other members of the family had also suffered from a similar disease.

In 11 cases, one or more of the infectious cases had preceded the onset of the dystrophic manifestations. In two the labor was said to have been difficult; in a third instruments had been used in the delivery; in a fourth the mother had suffered from dystocia in a previous labor, on account of the large size of the child; and in a fifth it was stated that the mother had been subjected to considerable anxiety during the pregnancy. In three cases there had been falls upon the back before the advent of the symptoms of the muscular trouble. The father of one patient had been alcoholic and his family tuberculous; the father of another had died in the sequence of an apoplectic attack. An uncle of another patient had been insane. The grandfather of one had been paralyzed. Of another patient one sister had died of meningitis, and one during dentition.

I desire to call attention to the contraction of the muscles of the upper part of the thigh noted in Cases XIX. and XX., when the sole of the foot was gently irritated, notwithstanding the absence of the plantar reflex. I have since observed a similar phenomenon under other conditions when the plantar reflex was not interfered with. I am as yet unable to say anything as to the frequency of its presence or as to its significance. It is probable that it is an entirely normal manifestation, and it may be that attention has already been directed to it.

In speaking to Dr. S. Weir Mitchell upon the subject of the muscular

[1] Pseudo-hypertrophic Paralysis. A Clinical Lecture, 1879.
[2] Dystrophia muscularis progressiva. Leipzig, 1891.
[3] Op. cit.

dystrophies, he raised the interesting question as to the occurrence of this group of disorders in colored persons and especially in those of pure, unmixed African parentage. It is known that chorea and locomotor ataxia are exceedingly rare in blacks, and it is probable that exophthalmic goitre and other diseases are so likewise. In none of the cases here detailed is any specific reference made to the race of the patients, and it is not unfair to assume that even those cases in which definite knowledge upon this point is wanting occurred in white persons, inasmuch as the matter of race has always received especial consideration at the Infirmary, and the physicians under whose care they were have no recollection of ever having seen a case in a colored person. The literature of the subject is quite enormous, and I have not taken pains to scour it; but nowhere have I seen any reference to the occurrence of the dystrophies in other than white persons, and my personal experience, together with information derived from a variety of sources, points to the rarity of these diseases in blacks. In the hope of determining this question, I addressed letters of inquiry to a number of neurologists and general practitioners in different parts of the country, whom I take the opportunity here of thanking for their courtesy in replying. I append extracts from some of the communications received:

Dr. Curran Pope, of Louisville, writes: "I have had almost daily contact with the colored race in dispensary practice, and . . . . they constitute about 65 per cent. of the people attending the clinics in this part of the country. I have never seen a single case of any of the muscular dystrophies in the full-blooded African or mulatto race. . . . . I have seen cases of every atrophy and dystrophy, especially the idiopathic, either in dispensary or private practice, but all have been in the white race."

Dr. Hugh Hagan, of Atlanta, Ga., writes that he has seen three cases of progressive muscular atrophy of Aran-Duchenne type in negroes, two pure Africans and one a mulatto. He states further that he has seen a case of upper-leg atrophy in a negro porter, and one of scapulohumeral atrophy in a farm-hand. He believed that the blood of the latter was not pure, although the patient maintained the contrary. Of the Erb type he has also seen one case in a colored child of undoubted syphilitic parentage.

Dr. M. S. Iseman, of Georgetown, S. C., writes that he can recall but one case of muscular atrophy in a negro, and that was evidently one of anterior poliomyelitis.

Dr. F. X. Dercum states that "I have never seen a myopathy in a colored subject. Muscular atrophy of spinal origin I have seen quite frequently."

Dr. C. W. Burr says: "I have seen one case of idiopathic muscular

atrophy occurring in a young mulatto boy. I have never seen a case in a pure black, but do not know whether it is really infrequent."

Dr. W. Peyre Porcher, of Charleston, S. C., expresses the opinion that "these neuroses are extremely rare in the negro race, although the true paralyses are by no means uncommon."

Dr. Landon Carter Gray states that he has seen only "isolated cases of muscular atrophies in colored persons, for we do not see much of this race in our clinics."

Dr. B. Sachs writes that he has "not seen a single case of muscular dystrophy in a colored person, nor have I any knowledge of its occurrence among persons of unmixed African parentage."

Dr. J. Hendrie Lloyd states that he cannot recall having seen examples of the muscular dystrophies in negroes. "My general impression is that these dystrophies are rare in the unmixed African races."

Dr. Charles K. Mills writes that he cannot recall any cases of muscular dystrophy in colored persons. "I am inclined to the opinion that these affections are extremely rare in the colored race."

Dr. M. Allen Starr writes: "I have never seen a case in a colored person."

Dr. William Osler does not remember any case of muscular dystrophy in a colored child.

Dr. Henry Posert, of Memphis, Tenn., writes that among the cases of atrophies and dystrophies that he has seen there has been none in an individual of pure or impure African parentage. "I am sure," he adds, "that if any cases had been in town or in the neighborhood I would most likely have seen them." Dr. Posert refers to a typical case of facio-scapulo-humeral atrophy (Landouzy-Déjérine) combined with pseudo-hypertrophic paralysis.

Dr A. Earle Boozer, of Columbia, S. C., writes that he has not had any experience with the muscular dystrophies, and inquiry among his local colleagues discloses the fact that they, too, have not observed examples of the disorder.

Dr. George J. Preston, of Baltimore, Md., is unable to recall any case of muscular dystrophy in a negro. "Like certain of the degenerative cord-diseases," he says, "it must be rare."

Dr. J. A. Witherspoon, of Nashville, Tenn., states that he is unable to find recorded in his note-books a single case of muscular dystrophy in the pure negro.

Dr. Rudolph Matas, of New Orleans, informs me that he has some remembrance of a case of pseudo-muscular hypertrophy in a negro child and one of general atrophy in an adult, but nothing that he could describe accurately. He goes on to say, further: "On the general basis

that the negroes are much less liable to nervous diseases than the whites, I would be inclined to believe that muscular dystrophies and atrophies are not so frequent among them as in the white population. As bearing upon this question I would quote the statistics on nervous diseases in the Charity Hospital. In the decennium 1884–1894 there were 4312 patients with nervous diseases in the in-door services of the hospital; of this total 77.9 per cent. were whites and 22.1 per cent. colored. The actual mortality was 18.46 per cent. in whites, and 33.89 per cent. in the colored. The decennial prevalence (ratio to hospital population) was 7.43 per cent. in whites, 4.95 per cent. in the colored, or 74 cases of nervous disease in 1000 white patients and 49 in 1000 colored. The decennial mortality was 1.37 per cent. in whites (14 : 1000) and 1.68 per cent. in the colored (or 17 : 1000)."

Of the pathogenesis of the muscular dystrophies we know absolutely nothing. As they manifest themselves almost invariably in the early period of life, the thought naturally arises that they may be due to some aberration in development. On the other hand, as the muscular affection is variable and widespread in distribution and progressive in course, it is not unreasonable to suppose that it may be dependent upon a general metabolic disturbance, such as may arise from derangement of some internal secretion. It is on account of the latter reason that the view promulgated by Macalister[1] is entitled to thoughtful consideration. This observer has formulated the hypothesis that all of the glandular structures of the body elaborate substances that exert a controlling influence upon the growth of individual tissues. In those morbid conditions characterized by hyperplasia or hypoplasia of a single tissue-element, there may be supposed to be an absence or a perversion of the secretion that physiologically controls the growth of that particular element. The occurrence of the muscular dystrophies corresponds in time with the period of functional activity of the thymus gland, and it is thought that premature cessation of the secretion of this gland may be responsible for the trophic changes that take place in the muscles. Upon the strength of this hypothesis, which, it must be admitted, is not without plausibility, the employment of thymus gland or of an extract of the gland has been recommended in the treatment of the muscular dystrophies. I am not aware, however, that this suggestion has been acted upon except in the one case in which Macalister himself pursued the plan I proposed, and the results of which are not stated. Lépine[2] has, however, employed thyroid gland in the treatment of two cases of muscular dystrophy, in both of which increase in muscular vigor followed, and in one also increase in the size of wasted muscles.

[1] British Medical Journal, No. 1684, p. 729.
[2] Lyon Médical, May 10, 1896, p. 35.

Tabular Summary of Cases.

No.	Name.	Sex	Age at application.	Age at first symptom.	Similar disease in relatives.	Nervous disease in relatives.	Previous disease of childhood.	Complicated labor.	Traumatism.
1	M.E.B.	F.	3½ yrs.	Birth.					
2	W. W.	M.	9 "	6 yrs.	.....	Father alcoholic.	Pneumonia, 4 years.		
3	A. H.	M.	11 "	8 "					
4	H. D.	M.	8 "	8 "	.....	.....	Scarlet fever, 4 years.		
5	F. U.	M.	11 "	5½ "	.....	.....	Scarlet fever, 5 years.		
6	L. S.	M.	10 "	2½ "	.....	.....	Measles, pneumonia, 2 years.		
7	J. P.	M.	22 "	7 "					
8	J. B.	M.	7 "	2 "	.....	.....	.....	Instrumental delivery.	
9	E. W.	F.	9 "	Birth.	.....	Uncle insane.			
10	C. S.	M.	9 "	1 year.	.....	.....	Measles.		
11	P. C.	M.	11 "	7 yrs.	Brothers.	.....	Pneumonia.		
12	A. C.	M.	9 "	7 "					
13	P. T.	F.	7½ "	1 year.	.....	Grandfather paralyzed			
14	C. C.	M.	8 "	3 yrs.	.....	.....	.....	Hard labor; large child; no instruments.	
15	J. R.	M.	10½ "	1½ "	.....	Sister meningitis; sister dead dentition.	Whooping-cough, chicken-pox.	Dystocia with another child unduly large.	
16	W.G.C.	M.	8¾ "	1½ "	.....	.....	.....	Difficult labor; no instruments; no injury.	Fall from coach at one year; no sequel.
17	N.M.J.	F.	16 "	12 "	.....	.....	Measles, 2 years: whooping-cough, 4 yrs; influenza, 12 years.		
18	J. W.	M.	24 "	5 "	.....	.....	Variola, 10 years.	.....	Fall from 2d story window in childhood.
19	E. J. R.	M.	19 "	5 "	Great-grand-uncle; grand-uncle.	.....	Erysipelas, 5 years.	.....	Fall on back at 5 years.
20	G. K.	M.	22 "	3 "	.....	Father, apoplexy	Chicken-pox, mumps, measles.	Mother suffered anxiety during pregnancy.	

REVIEWS.

THE ELEMENTS OF CLINICAL DIAGNOSIS. By PROFESSOR G. KLEMPERER, Professor of Medicine at the University of Berlin. First American edition, from the seventh (last) German edition. With sixty-one illustrations. Authorized translation by NATHAN E. BRILL, A.M., M.D., Adjunct Attending Physician, Mount Sinai Hospital, New York City, and SAMUEL M. BRICKNER, A.M., M.D., Assistant Gynecologist, Mount Sinai Hospital, Out-patient Department. New York: The Macmillan Company; London: Macmillan & Co., Ltd., 1898.

ONE of the most popular compends on clinical diagnosis in Germany has been the little work of G. Klemperer, of Berlin, the first edition of which appeared in 1890. The seventh German edition has been translated into English by Brill and Brickner, preserving very nearly the form of the German work as regards size, shape, type, figures, etc., so that it is in reality a reproduction of the original. Even the Latin terms for diseases are retained.

Whatever may be the views of the teacher or the practitioner as to the value of these compends there can be no question as to their popularity among students. A really good condensation of the main facts of medicine systematically arranged, as is the case in this book, meets with the student's approval, and the book sells.

The great marvel in this book is not so much that a great deal has been crowded into a small space, as that it has been done so systematically.

The great trick in packing a trunk is not alone to know what to leave out, but also to know in just what way to put in the necessary articles so that they shall occupy the least possible space without injury to themselves or their neighbors. One marvels at the compactness and neatness with which Klemperer has tucked in his facts, and only finds an explanation of his ability to do this work so well when one remembers that he has for some time been engaged in the work of teaching small classes. He has learned what facts students need; he knows where the student is likely to fail and what points need emphasis. It is, therefore, valuable as a book to be consulted in reviewing for examination, or for hints as to the nature of some obscure concrete case on which the student is working, or to give him a rapid knowledge of the place occupied by a certain disease in its relation to other diseases; in other words, to afford a quick general survey of the subject.

The great danger from the use of these books is that from their very nature statements must be brief and often dogmatic. The student has impressed upon him the typical cases. The numerous exceptions puzzle him when he meets them in practice, because they do not fit in with the arbitrary statements of the compend. Many of these statements, if taken literally, are not true: "Systolic murmur over the aorta is diagnostic of aortic stenosis" (p. 169). "Actual murmurs, audible without pressure, heard over peripheral arteries, prove the presence of an aneu-

rism, and are usually palpable" (p. 171). "A systolic murmur over the mitral denotes insufficiency of the mitral" (p. 169).

These are all true within certain limits. But if these sentences are memorized by the student and looked upon by him as infallible guides, he will commit errors in diagnosis, and will find at some autopsy, to his astonishment, that the systolic murmur over the aorta did not mean any stenosis of the aortic valve.

While Klemperer may have some good reason for believing that auscultation in mitral insufficiency is practised in the second left intercostal space (p. 168), and that pericardial friction-sounds are not synchronous with the heart's beat (Herzaction) (p. 170), and that the pulse is slow in mitral stenosis (p. 178), these statements, at variance with what is generally taught by good clinicians, deserve explanation that would take but little space and would enable the student to realize that on some points in medicine authorities may differ. Several other dogmatic statements that, if not erroneous, at least call for explanation, are found in other parts of the book than in the chapter on the Heart, from which we have made our selections.

Brief portraitures of all the important diseases, condensed discussions of such topics as physical diagnosis, pulse, fever, examination of secretions, blood, urine, the animal and vegetable parasites, the disorders of metabolism, etc., are as clearly given as could be expected in so small a space. Three pages on the use of the Röntgen ray as a diagnostic aid are appended.

For a small compend of clinical diagnosis the work is an excellent one, and the translation has been well done, the terse sentences of the original with its omission of unnecessary adjectives, articles, and verbs being preserved in the English. J. B. H.

DIE TECHNIK DER SPEZIELLEN THERAPIE FÜR AERZTE UND STUDIERENDE. VON DR. F. GUMPRECHT, Privatdocent in Jena. Mit 181 Abbildungen im Text. Jena: Gustav Fischer, 1898.

TECHNICS OF SPECIAL THERAPY. By DR. F. GUMPRECHT.

THE great development of applied therapeutics in Germany in recent years had as a notable evidence the comprehensive handbook of Penzoldt and Stintzing, in six large volumes, now in its second edition. The present work is still more noteworthy, partly because of its intrinsic value, partly because it is the product of one who has hitherto been known only as an accomplished and energetic investigator in many departments of internal pathology and diagnosis. Under such men as Fürbringer and Stintzing he has been able to acquire a valuable experience in the treatment of disease, which saves this book from being merely a well-arranged and very full compilation.

The title is difficult to render into English, the original being misleading when we remember there are technics of purely medicinal treatment, with which the volume before us has nothing to do. The author says that his aim was to limit the subject on the one side where the work of the nurse, on the other where that of the specialist begins. We cannot do better than continue the quotation: "Difficult and complicated manipulations like laryngeal operations, uretero-cystoscopy, gastroscopy, and pure cutting operations are omitted. So we have

intubation, but not tracheotomy; pleural drainage, but not costal resection; urethral divulsion, but not urethrotomy." The topics include the mechanical treatment of the mouth, nose, pharynx and larynx, œsophagus, stomach, intestines and rectum; thorax; abdomen; puncture of special abdominal organs; lumbar puncture; nerve stretching; suspension; catheterism; treatment of strictures and enlarged prostate; local treatment of the urethra; subcutaneous drainage in anasarca; bleeding, transfusion, and vaccination. These are described with great detail, with the single exception of vaccination, which the author no doubt thinks is sufficiently well known, as it no doubt is in Germany. As an example of the plan followed, an outline of the chapter on the Stomach may be given. In this are described the limits and objects of instrumental treatment; anatomic data; historical note; lavage and its indications, relative and absolute; method; accidents to the apparatus and to the patient; stomach douche; the questions as to time, frequency, and duration of lavage; electric treatment; massage; special methods of treatment for the pylorus. In every detail we discover not only a very full command of the literature, especially German, but also a very well-digested personal experience. The style is clear, the mass of facts often relieved by incidents in practice or personal reminiscences. The work, therefore, fills a unique place in medical literature, and must be especially valuable to those who have to treat all kinds of diseases, though even specialists will find the book useful for easy reference. Many physicians fail to carry out such operations as are here described because they do not know how. It is true that such things should be learned in hospitals, but they can be learned from books if properly read, and this one is superior to any we know for that purpose. In general all the methods recommended are such as are in common use among experienced physicians everywhere, but there are some things that show the independence of the German and his ability to get along with simple apparatus. For example, the author recommends the removal of obstructions in the stomach tube by blowing, telling how to avoid being surprised by the patient vomiting during this procedure. We think one might reasonably object to being considered squeamish ("heikel") because he does not care to blow up the colon by the mouth as practised by Furbringer. The hand-bulb is surely more convenient. In bleeding, the author recommends the old operation with the lancet, instead of the time-consuming operation by laying bare the vein usually practised by those educated in modern surgical technique.

The typographical arrangement of the book is excellent; useful bibliographical lists are appended to each section, and a well-arranged index completes what must be pronounced a most admirable guide to the art of medicine. G. D.

THE GENESIS AND DISSOLUTION OF THE FACULTY OF SPEECH. A Clinical and Psychological Study of Aphasia. By JOSEPH COLLINS, M.D. Awarded the Alvarenga Prize of the College of Physicians of Philadelphia, 1897. New York: The Macmillan Company; London: Macmillan & Co., Ltd., 1898.

THE author of this work has studied carefully the literature of aphasia, and has presented the most recent views held by some of the

best writers on this subject. The influence of the teaching of Dejerine is clearly seen throughout the book; so much so that one would be inclined to think that it had been written by a pupil of the distinguished French master. It may, therefore, be looked upon as the best exposition in English of this French school, and yet we should be sorry to give the impression that it is a mere copy. Collins has thoroughly digested all he has read, and has given us much that is original. No work on aphasia can ignore the study of those who have preceded us.

The definition of aphasia as a "term used to indicate any disturbance or perversion of intellectual expression" is certainly far-reaching and misleading, and includes lesions of the peripheral neurons. Many will object to classifying disturbances of speech, such as are seen in bulbar paralysis, under aphasia. We find Collins himself, in describing Elder's case, saying "there was no aphasia," although the patient had, apparently, "incapacity to enunciate words," and it would have been better had the foot-note on page 155, in which the views of the author are more clearly stated, appeared nearer page 14.

We can state in a few words the position taken by Collins in his conception of aphasia, and it will be seen at a glance how closely he follows Dejerine. There is a speech-zone about the left Sylvian fissure in right-handed persons, comprising the foot of the third frontal, the posterior part of the first temporal, and the angular gyrus. Lesions of any part of this zone affect speech as a whole, with special manifestations according to the area diseased. A centre for acoustic images of words is found in the first temporal convolution, one for visual images of words in the angular gyrus, and one for articulatory kinæsthetic images in Broca's area. Destruction of the fibres leading to or from these centres causes subcortical or pure forms of aphasia. A special centre for writing does not exist. Collins reviews the testimony in favor of this centre, and rejects it. Broca's area is not a motor region. In it are stored the articulatory kinæsthetic images; it is a controlling centre for the cortical motor areas, but the cortical representation of the muscles of speech is elsewhere. The whole speech-zone, therefore, according to Collins, is sensory. Vision is represented in the occipital lobes, but a higher centre of vision is located in the left angular gyrus.

We fully agree with the author when he says that the terms "ataxic aphasia" and "amnesic aphasia" are indefinite, and should, therefore, be avoided. We agree with him also in the statement that the integrity of the frontal lobes is essential to the full exercise of complex thought; but might not the same be said of any of the cerebral lobes? The reviewer has found the entire left occipital lobe destroyed in a feeble-minded individual, and believed that this stood in causal relation to the feeble mentality.

The chapter on the History of Aphasia is short, as this is not the most essential part of the work, but we should have been pleased to find a little more attention paid to certain writers.

We fear that Dejerine's views, as understood by us, are very different from those attributed to him on page 101. The inferior pediculo-frontal fascicle seems to us a very unfortunate name. Presumably, the inner bundle of the cerebral crus is meant, but this passes through the cerebral peduncle, and possibly terminates in the pons. We might as well call it the fronto-capsular bundle. Must this tract "go"? Are all anatomists agreed on this point? The reviewer, from his microscopical

studies, is convinced that some of these fibres arise anteriorly to the Rolandic convolutions, and he is not alone in holding this opinion.

Collins reports a number of interesting clinical cases, some with necropsy, but none with microscopical examination. These cases have been studied clinically with much care.

The case reported by Banti and quoted on page 167 was probably one of pure or "subcortical" motor aphasia, due to a cortical lesion, and, since the recent publication by Dejerine and Sérieux of a case of pure word-deafness due entirely to cortical lesions, we may well ask whether the term "subcortical" will not have to be abandoned.

We find the influence of Dejerine again in the statement that a patient with cortical motor aphasia is agraphic, except in writing from copy, and is dyslexic.

Dynamic aphasia is always of the greatest importance, and the case reported on page 354, due to bromide intoxication, is one of considerable interest. Our knowledge of aphasia as a complication of Raynaud's disease is increased by the report of a case.

We are not quite prepared to accept the statement that "tumor presents the ideal lesion" in localizing speech centres. Tumor causes many symptoms by pressure, and this truth has been recently clearly shown in a case reported by Oppenheim, in which symptoms of aphasia were dependent on the position of the patient. A tumor was found at the necropsy. Areas of softening are more focalizing in their symptoms.

We echo the author's words when he speaks of the necessity of microscopical study in every case. He who depends on gross appearances alone in studying the nervous system will be led into great error. Lesions of vast importance are sometimes detected only with the microscope.

The directions given on page 380 are not in accordance with the teaching of Dejerine. Redlich's case was one of lesion in the occipital lobe, and the sections were frontal ones.

The possibility of complete restoration of the cortex supplied by the Sylvian artery after it has been injured by occlusion of this vessel in childhood must be regarded with some scepticism. Extensive sclerosis of this part of the cortex has been found after such lesions.

Although the author believes that theoretically there are grounds for the belief in conduction aphasia, he is liberal enough to acknowledge that all the cases which have been reported may be looked upon as manifestations of sensory aphasia.

We have mentioned merely some of the most striking features of this interesting and valuable book without attempting to do full justice in a brief review. W. G. S.

THE ETIOLOGY AND PATHOGENESIS OF YELLOW FEVER. By Prof. G. SANARELLI. Il Policlinico, 15 Agosto, 1897.

THIS is an article of 100 pages with several colored plates and photographs, and gives the detail of the investigation of the author on yellow fever. The work is divided into two parts. In the first part he considers:

1. The existing knowledge on the etiology and pathogenesis of the disease when he began his investigations.

2. The isolation of the specific organism.
3. The character of the anatomical lesions produced in man by the disease.
4. The morphology and biology of the bacillus icteroides.
5. The comparative pathology—results of inoculation.

In the second part of the article he gives the results of his studies on the toxin produced by the bacillus, and considers the general pathology of the disease, modes of infection, etc.

Yellow fever has long been recognized as a disease of specific charac ter, but its etiology has not been known in spite of the many attempts which have been made to discover a specific organism in connection with it. The description of the clinical features of the disease does not differ from that given by Sternberg and others. The investigations of the author were made partly at the quarantine station at Montevideo, and partly at Rio Janeiro. Thirteen cases were examined, and in seven a peculiar bacillus was isolated which he regards as the cause of the disease.

In the second case examined this organism was found in the blood taken before death, and in the blood, spleen, liver, lungs, urine, and bile at the autopsy. In all these places the organism was found in pure culture, but in the kidneys and bronchi it was associated with the colon bacillus. It was rarely found in pure culture, and most often was associated with some of the pyogenic cocci or with the colon bacillus. In the seventh case the cultures gave an abundant pure growth of the streptococcus pyogenes in all the organs. He thinks the reasons why this bacillus has not been discovered before are the small numbers in which it is found and the probability of secondary infection from the alimentary canal by other organisms, which may obscure the presence of the bacillus icteroides. The most common of the secondary infections are by means of the colon bacillus, the streptococcus or the staphylococcus aureus. These organisms may produce a true septicæmia and themselves lead to the death of the patient. From the result of cultures it is shown that the bacillus is not contained in the intestinal canal, but in the blood, by means of which it may be carried into all of the organs. The lesions found in the organs consist of extensive and diffuse degeneration, and are due to the action of toxins produced by the bacillus. The most profound lesions were found in the liver, which may present but little evidence of alteration on macroscopic examination. Microscopically there is extensive fatty degeneration, followed by complete destruction of the cells. The degenerative lesions in the kidney, though marked, were not so extensive as in the liver. He was not able to demonstrate the bacillus in sections of the tissues made in the ordinary way, but on keeping large portions of the tissue in the thermostat for twenty-four hours, thereby allowing the few organisms present to undergo a further development, he found them in fair numbers. The lesions in the stomach consist in mucoid degeneration and desquamation of the epithelium. The black vomit is a hemorrhage which takes place from the exposed vessels.

The bacillus icteroides is a small rod, rounded at both ends, ciliated, 2 to 4 micromillimetres long, and two or three times as long as it is wide. It has no peculiarities of staining. It grows readily on all of the artificial media, and growth may take place at a temperature of 20° C. The most characteristic cultures are those on gelatin. After twenty-

four hours the colonies appear under a low power as small transparent or slightly granular masses very similar to leucocytes. When the colonies are abundant and growing within the medium, they remain small and become converted into small opaque points. On the surface they grow rapidly and preserve their brilliant, slightly granular appearance. One of their most characteristic features is the gradual appearance of a small dark spot in the centre. After eight, ten, or twenty days the colonies undergo a slow and gradual transformation. They assume a yellowish or brownish tint, and fissures running to the centre of the colony or concentric lines appear in them. The bacillus produces abundant acid.

The organism was pathogenic to all animals inoculated. The most important inoculations were made on dogs, and the lesions were very similar to those found in man. In some cases death took place from acute septicæmia in twelve to twenty-four hours, and in others the disease ran a more protracted course. Autopsies showed sometimes acute gastritis, with necrosis and exfoliation of the lining epithelium, laying bare the bloodvessels from which hemorrhage took place. The spleen was usually found enlarged. Microscopically there was the same fatty degeneration of the liver, with the destruction of cells, which he found in man. The blood of the animal always contained a large amount of urea; in some cases as much as 4 per cent. He considers the lesion to be the result of a toxin circulating in the blood, and the intestinal lesions are very analogous to those which may be produced by the poisons of certain bacteria and by cyanide of potash. The excessive amount of urea in the blood is due to the degenerative lesions of the kidney. The bacillus produces not only a general intoxication, but its toxins have an elective affinity for certain organs, as the kidneys, the digestive canal, and the liver. Many of the symptoms are due to uræmia, which comes from the anuria, the result of the degeneration of the kidneys. The hemorrhagic character of the intestinal lesion is due to the action of the poison, which produces profound alterations in the bloodvessels. The lesions produced in the mouse, the guinea-pig, and the rabbit are of the same general character, and proved fatal in the first in five days, in the second in six to eight days, and in the third in five days. In the dog the clinical features were frequently almost exactly the same as those seen in man, and consisted in vomiting, hæmatemesis, hæmaturia, albuminuria, gastro-enteritis, nephritis, jaundice, profound fatty degeneration of the liver, uræmic intoxication, and secondary infection. There are three different properties of the virus. The first he calls the steotogenous, which produces the fatty degeneration; second the hemorrhagic; and, third, the emetic property. He made a number of experiments with filtered cultures of the organism, and injected a small amount of the toxin into five men. The symptoms produced were analogous to those of true yellow fever. In one of them he obtained by aspiration some of the parenchyma of the liver and kidney, and found in the cells the same degenerative lesions which he observed in the definite disease. He thinks the jaundice in yellow fever may be of hæmatogenous origin, because he was never able to find bile pigment in the urine nor in the fluid of the tissues. It is probably due to destruction of the red corpuscles of the blood accompanied by hepatic insufficiency. The nephritis and consequent uræmic intoxication is the result of a specific action of the toxin on the renal parenchyma.

He is not certain as to the manner in which the organism enters into the tissues, but he thinks it may do this in a number of ways. Most of the facts seem to favor the transmission of the organism by means of the atmosphere. It is interesting to find that moulds favor in an inexplicable way the growth of the organism, and to this is probably to be attributed the influence of dampness in favoring the spread of the disease. In some cases the disease seems to appear more frequently in the houses on the north side of the street, these houses being more damp and favoring the growth of moulds. The organism appears easily killed by heat. It is destroyed by one minute exposure to a temperature of 65° and by a three-minute exposure to a temperature of 60° moist heat. It is killed in dry heat in ten minutes at a temperature of 100°, and at once at a temperature of 120° to 125°. It stands dryness extremely well. Cultures are quickly sterilized by direct sunlight.

The article impresses one as a careful piece of work by an experienced bacteriologist. The pathological and anatomical descriptions of the lesions are not as accurate as they might be. He has overlooked the fact that the most important lesions in the liver consist in a general and diffuse necrosis of the cells, occurring principally around the central veins of the lobules, and which may occur with but little fatty degeneration. His conception of the general pathology of the disease and the relation of the organism to the lesion is certainly very reasonable. Whether or not the definite etiology which he claims be confirmed by future investigations, the work constitutes a valuable addition to our knowledge of this very obscure disease. W. T. C.

Constipation in Adults and Children. By H. Illoway, M.D., Formerly Professor of the Diseases of Children, Cincinnati College of Medicine and Surgery, etc. Pp. xv., 495. New York: The Macmillan Company, 1897.

This work deals especially with habitual constipation and its treatment by mechanical methods. Commencing with the anatomy and physiology of the intestine, the author takes us over the etiology, symptomatology, diagnosis, prognosis, and consequences of this symptom. Under treatment massage is given a prominent place, and is fully described and illustrated by various figures of more or less artistic merit. Hydrotherapy and electricity receive suitable attention. Medicines are discussed in a brief chapter. Next conditions other than atony are taken up and full directions presented. A chapter devoted to formulas ends the first part, which deals with this symptom as found in adults. About a hundred pages are occupied with constipation in infants and children. The book is well arranged, thoroughly classified, and shows an extensive acquaintance with the existing literature both medical and surgical. For all that mechanical methods are those upon which the author lays great stress, nowhere does he take the extreme position that drugs are useless. In fact, he directly contradicts those who pander to popular taste in decrying the use of drugs when he expresses his scepticism that purgative-taking is a cause of constipation (p. 101). Even further than this, the extensive formulary which

he presents demonstrates his belief that if drugs alone cannot afford relief, they certainly are necessary adjuncts to massage, graduated resistive movements, and a regulated diet. For the last there are found excellent directions. He differs with some of his contemporaries in believing that the closet should be visited at least once daily shortly after a meal (not of necessity after breakfast), thus relying upon an "irregular regularity" (p. 187) to secure satisfactory evacuation. He also condemns the fashionable practice of drinking hot water on rising in the morning (p. 193). The points for criticism lie in faulty orthography in pharmacopœial terms and designations, and in peculiar abbreviations. These may be overlooked when we consider the thoroughness with which he has treated—we might say, exhausted—the subject, and the many practical points which are so frequently encountered.

R. W. W.

A System of Medicine. Edited by T. C. Allbutt, M.D. Vol. IV. Diseases of Obscure Causation; Alimentation and Excretion. The Macmillan Company, 1897.

This volume is the most interesting one of the series which has yet appeared, and the high excellence of the papers contributed makes the *System* one of permanent value. We cannot mention all the well-known men who have written articles for this volume, but among them are such authorities as Drs. Church, Cheadle, Garrod, Lauder Brunton, Fenwick, Hale White, Sir William Roberts, Mr. Bowlby, Mr. Treves, the editor, and many others. Vol. IV. begins with General Diseases of Obscure Origin, including Rheumatism and Rheumatoid Affections, Rickets, Gout, Diabetes, etc.; then follows a chapter on Diseases of Alimentation and Excretion, which, beside comprising affections of the entire digestive tract, includes also Diseases of the Peritoneum, Subphrenic Abscess, etc.

The general arrangement of the work is excellent, and the study of the maladies is made clearer by special introductions on the general pathology of digestion and secretion, and on the physiology of fecal evacuation. There is a chapter on Shock and Collapse, which follows the paper on Secretion and precedes the one on Diseases of the Mouth. Dr. Cobbett has discussed the two conditions in a clear and able article, but we do not quite see the justification of its introduction at this place. Such articles as do not deal with disease, it seems to us, would be better grouped together in one volume by themselves. Dr. Playfair's paper on Abdominal Diseases from a Gynecological Standpoint is also out of place, from our point of view.

We do not wish to find fault with the individual contributions, but it appears more markedly in this volume than in others, that not sufficient attention is given to the relative length of the papers; for instance, the longest one in this volume is by Mr. Treves, on Intestinal Obstruction, which covers eighty-five pages; the shortest, three by Mr. Bowlby, on Osteitis Deformans, Acromegaly, and Hypertrophic Pulmonary Osteoarthropathy, to which but six pages are given. The condition of intestinal obstruction is one which concerns the surgeon more than the physician in the great majority of cases, and, as its interest is very

largely in the anatomical findings, the reason for the introduction of so long a paper on this disease is not self-evident. It is, however, an article of intrinsic value, from the extreme care with which each possible form of obstruction has been described.

Perhaps Mr. Bowlby is right in not devoting more space to three diseases of such obscure origin as those mentioned, but we feel as if they were treated, although succinctly, in too cursory a manner. Massalongo has lately expressed a view that in hypertrophic pulmonary osteo-arthropathy the changes in the bones have nothing whatsoever to do with the pulmonary complaint, but are due to other causes, and that rheumatism, syphilis, etc., may be factors in the causation. When we consider that all the pulmonary changes with which the disease is associated are much more frequently found without any alterations in the long bones, it is only fair to admit that Massalongo's theory may be the right one, and that the name "secondary hypertrophic osteo-arthropathy," which he suggests, is better than the one by which the disease is commonly known.

Diseases of obscure causation offer many points for differences of opinion, and for this reason it is, perhaps, not worth while to take up any of the papers at length and discuss them. It is noteworthy, however, that Dr. Brunton, in his article on Constipation, says almost nothing on the value of aperient waters, nor mentions the use of a cigar after breakfast. Dr. Spender, in discussing the origin of rheumatoid arthritis, lays much weight on its association with tuberculosis. At the British Medical Association, held at Montreal in 1897, the subject of rheumatoid arthritis came up for general discussion, and Dr. James Stewart, in his address, referred to the relationship of these two diseases, but did not find that close causal connection on which Dr. Spender seems to insist. Dr. Stewart said "indirectly a tuberculous tendency may, by lowering the resistance, tend to bring about a rheumatoid arthritis, and it is only in this sense, as I understand it, that there is a connection between the two diseases." This opinion seemed to meet with common agreement of those present at the discussion, and it is as yet doubtful whether any closer connection between the two diseases does exist; it seems improbable.

We feel justified, in concluding this review, in stating that this is the best *System* at present being published, and that it is one which every student of medicine should possess. The length of some articles and the arrangement of others may not be the best, but taken as a whole the work is one of very high excellence, due both to the ability of the editor and to the first-rate selection of contributors. R. N.

HANDBOOK OF MATERIA MEDICA, PHARMACY, AND THERAPEUTICS. By SAMUEL O. L. POTTER, A.M., M.D., M.R.C.P. Lond., Professor of the Principles and Practice of Medicine and Clinical Medicine in the College of Physicians and Surgeons of San Francisco; Medical Superintendent of St. Mark's Hospital. Sixth edition fully revised, and greatly enlarged. Pp. xv., 900. Philadelphia: P. Blakiston, Son & Co., 1897.

THIS edition comes to us with an increase in size commensurate with the additions to the resources of the physician, with new sections and

others extended and rewritten, so that the volume becomes a fair presentation of the subject as it stands to-day. In the portion devoted to therapeutics there is to be found still greater revision and elaboration. Of special interest are the articles upon toxins, antitoxins, and animal extracts, in which the author has presented a careful digest of current literature and reached an opinion as regards their value which can be accepted as one that will not be reversed during the next decade. In this he has separated the anticipation from the realization, the theoretical from the practical, and the presumption from the proof. An interesting feature is the insertion and brief description of a considerable number of unofficial remedies of greatly varying importance and usefulness which are constantly met with in medical journals. Concerning some he speaks in no uncertain terms: for example, concerning aseptolin (p. 78), "There is nothing original in this treatment, it being a repetition of the phrenic acid injection of Declat combined with the pilocarpine treatment of phthisis announced about two years ago as the 'discovery' of Dr. Waldstein. It is now going through the usual puffing methods of trade promotion, and the few independent reports upon its use show no special merit for it in these diseases" (tuberculosis, malaria, and other diseases due to germ infection). More frequent examples of frankness on the part of therapeutic experts would prevent a large number of practitioners from becoming dupes. In Part III., Special Therapeutics, sandwiched between Surgical Shock and Sneezing, we find a short article on sick-room. It is a very sensible plea for, and description of, a room to be set apart in every home. But if the author believes that "the possession of such an appendage to the home would enable the owner to defy the mandate of the health officer in regard to removing an inmate thereof," he fails to appreciate the crass ignorance, gross contempt for law, and innate venality of these officials as they are commonly met with. A knowledge of their cupidity would be of more importance than the establishment of a sick-room. The appendix contains a very complete list of contractions and Latin phrases, various formulas of solutions for hypodermatic use, patent medicines, tables of differential diagnosis, clinical examination of the urine, weights and measures, and other matters—truly a miscellaneous collection. The treatment of poisoning has been thoroughly revised and extended, leaving this subject in a very satisfactory condition. We have, in reviewing former editions, objected to the alphabetical arrangement of the materia medica, for the reason that the subject becomes solely a matter of memory on the part of the student. We find each year, in examination of candidates for hospital positions who have been graduated from different schools, that materia medica is less thoroughly learned. Whether the reason for this is the indifference or faulty methods of teachers, press of other and more attractive studies, or the dictionary text-books, we are not prepared to state; of the fact we are certain. On the other hand, the very great excellence of the section devoted to special therapeutics, which is so far in advance of the usual index of diseases that the work is of especial value to the junior practitioner, and in this lies, we believe, its value, which has been demonstrated by the publication of succeeding editions. Further, its well-considered optimism appeals to the faithful practitioner, while the frankness and clearness in style encourage him in his reliance upon its teachings. R. W. W.

PROGRESS

OF

MEDICAL SCIENCE.

THERAPEUTICS.

UNDER THE CHARGE OF

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The Treatment of Consumption.—Dr. Samuel Wilks has adopted a uniform treatment of this disease. In the first place, if a person is pursuing any course of life in which disease of the lungs is developing, that course must be altered. The remedy is not medicine, but fresh air; probably the very best air is to be found in the highlands of a southern climate. How far low barometric pressure is beneficial has not yet been proved, although it is likely of some importance. Sunshine quite possibly may be one of the conditions favoring recovery; it may be directly destructive to bacilli, as the warmth and the beauties of the landscape brought out by it indirectly excite the interest of the patient and revivify his nervous system.—*The Practitioner*, 1898, No. 360, p. 571.

Dr. James E. Pollock states that the communication of tuberculous disease by contact with or breathing the same air as persons suffering from that affection has been thoroughly investigated at the Brompton Hospital for Consumption, which affords the most abundant material. The possibility of its production by inoculation of a healthy animal with morbid matter has been proved beyond question. That, however, is a very different matter from the possibility of tuberculosis being conveyed by mere contact, however close to a patient so affected. The investigation covers a period of thirty-seven years. Of resident medical officers there were four; none had phthisis. Of about one hundred and fifty house physicians, eight became consumptive; in only one was the disease contracted in the hospital. Of six matrons, none had phthisis. Of one hundred and one nurses, one only had phthisis while in the hospital; three died of this disease after leaving the hospital. Of dispensers there were twenty-two: three died of phthisis. One was intemperate, one took ill two years after he left, and the remaining one died in the hospital. Of physicians and assistant physicians there were twenty-nine, of whom one died from phthisis. There were no deaths from

phthisis among the gallery maids, who sweep and clean floors several hours daily, nor among the secretaries and clerks. This evidence negatives the idea of phthisis being an infectious disease under such circumstances as being grouped in a hospital, breathing the same air, and living under the same conditions as others similarly affected. Later investigations have proved that consumption is communicable, although not by personal contact, and that its existence depends upon its transmission from one tuberculous individual to another. Such transmission is not aërial, but resembles more the process of inoculation. A certain amount of diseased product must enter into the animal economy, whether by the lung or the intestinal tract. It is recognized that the sputum of tuberculous patients, if dry, may be such an agent, and that the flesh and milk of tuberculous animals may convey the poison. In accordance with these views the sputum and excreta of consumptive patients are received into vessels containing a 20 per cent. solution of carbolic acid, and, together with the general refuse of the hospital, are consumed in an automatic destructor. All soiled linen and handkerchiefs are boiled, and all cups, spoons, etc., used by patients are disinfected in boiling water. The decrease in the death-rate must be attributed mainly to the improved drainage of the hospital, more cleanly habits, removal of insanitary surroundings, better dwellings, and a higher status of comfort in the lower classes.—*Ibid.*, No. 360, p. 609.

Dr Hector Mackenzie presents a thoroughly sensible article upon the medicinal treatment. All that we know of the disease is not very encouraging to the hope that an efficient antitoxin will be found. So far from immunity being acquired from the successful passing through the disease, the reverse may be asserted—namely, that susceptibility is increased once the bacillus acquires a successful footing in the body. Cod-liver oil has played an important part in the treatment for half a century. Although it is one of the best remedies which we possess for chronic cases, it is probably less in favor than formerly. As for creosote, while few are prepared to state that it has any specific action, most will admit that it has very valuable properties. Various preparations and derivatives of creosote have been found useful. Arsenic, or the arsenical mineral-water of Mont Dore, has been employed from very early times, and few drugs are believed to be more useful at the present day. The paper concludes with a brief consideration of the treatment of fever, night-sweating, hæmoptysis, and cough.—*Ibid.*, No. 360, p. 687.

The Open-Air Treatment of Pulmonary Tuberculosis.—Dr. C. Theodore Williams quotes Brehmer's laws: (1) That the sanatorium, to avoid dust, should be away from public traffic, and (2) that the air should be dry and aseptic, and yet there should be abundant rainfall. The leading principle is that the consumptive should pass the greater part of his time in the open air, protected from the weather, and, as a rule, in the prone position, and that at night he should sleep with windows open. This can be carried out in covered balconies or terraces suitably arranged. To resist cold the prone position is far better than sitting, probably on account of the equalizing effect it has on the circulation and the less strain which it exerts on the heart. Degrees of cold can be endured when lying down which would

cause chill in a sitting posture. This life in the open air hardens the invalid against fresh cold, increases the appetite, promotes sleep, reduces night-sweats, and, most important of all, lowers pyrexia. The objections to the reclining system are that, in advanced disease, it is not well adapted for cleansing cavities by expectoration, and, in incipient disease, without exercise, it is generally impossible to maintain the muscles and functions of the body in proper order. Opinions vary as to the necessity and extent of exercise. The dietetic treatment is that of stuffing with a rich and varied diet, including much meat, milk, fatty and farinaceous foods, and vegetables, given in large quantities at a time, with, in most cases, moderate intervals between meals, but sometimes very frequently. If the open-air treatment is to be introduced into any country, it must be modified, if it can be done without sensibly impairing its utility, to suit the spirit and habits of the people, and this can be done in country or even suburban houses.—*British Medical Journal*, 1898, No. 1951, p. 1309.

Dr. Arthur Ransome points out that neither a particular latitude on the earth's surface nor any great altitude can be considered necessary in establishing a place to carry out this treatment. Climate may, and does, prolong the existence of the phthisical, either by modifying nutrition and vitality in the mass, or by lessening the activity, perhaps warding off the occurrence of certain secondary states (bronchitis, hæmoptysis), which might of themselves have proved the real causes of death. No climate will stamp out phthisis, wherever acquired, either in the individual or in the stock. Of the results obtained, seventeen patients treated at Bournemouth have furnished the data. All were tuberculous, as shown by the finding of bacilli, save two. One of these did not expectorate; the other was a child, aged four years, with signs of a cavity at the apex of a completely consolidated lung, who completely recovered. Of these, two gained twenty-eight pounds in weight; one nineteen; nine others from five to twelve; one was stationary, and of four the weight was not taken. In seven the disease is now quiescent, and has been for periods of from one to two and one-half years, and they may be classed as relative cures. Five others are now quiescent, and have been for from four to six months, although they must be regarded as still under suspicion. One has improved, and three improved for a time, then relapsed, and the disease is now progressing. These patients show results which are inconclusive, but seem favorable to more prolonged trial of the system.—*The Lancet*, 1898, No. 3902, p. 1603.

Oil of Cloves in the Treatment of Pulmonary Tuberculosis.—Dr. H. A. Hare, in an address before the Medical Society of the State of Ohio, calls attention to this remedy, used both by the mouth and hypodermatically, for the purpose of decreasing cough and expectoration in advanced pulmonary tuberculosis. If given hypodermatically, five drops are mixed with thirty to sixty minims of the best French olive-oil, sterilized; considerable local pain is produced for a few minutes, but soon the anæsthetic effect of the oil asserts itself, and the relief from cough more than repays for the pain. The injection is given once daily into the loose tissues of the back. Internally, it is best given after meals in five, ten, to even fifteen-drop doses, but cautiously, as these may disorder the stomach. Not only are the

cough and expectoration greatly decreased, but the sweat and hectic fever are generally modified to very great extent.—*Therapeutic Gazette*, 1898, No. 5, p. 289.

How to Administer Creosote.—Dr. Charles Wilson Ingraham gives his patient an empty twelve-ounce bottle, in which four drops of beechwood creosote of a superior quality are placed. The second day five drops are put into the bottle, and so increased one drop each day until fifteen drops are taken each day. Now one drop is added every second day until the limit of twenty drops is reached. Later the dose is increased at the rate of one drop every third day until twenty-five drops per day are taken, and this must not be exceeded. After the prescribed daily amount of creosote is placed in the empty bottle it is filled with fresh water and the entire contents taken in equally-divided doses at regular intervals, making at least six or eight doses daily. The bottle must be thoroughly shaken before each dose. In this way the irritation of the throat so generally met with in pulmonary tuberculosis is directly relieved, the expectoration promoted, and the irritating cough benefited.—*Therapeutic Gazette*, 1898, No. 5, p. 299.

Thiocol.—Dr. C. Schwarz recommends this substance, which is a potassium salt of guaiacol sulphonate, as a new remedy in the treatment of tuberculosis. This occurs as a fine white powder, at first of a somewhat bitter, later, sweetish taste, and contains about 60 per cent. of guaiacol. Its advantages, as claimed, are its absence of odor, great solubility in water, absolutely unirritating properties as regards mucous membranes, and its ready absorbability. This is given in from 150 to 220 grains daily. It increases the appetite and general strength, the patient gains weight, the cough is lessened, the expectoration ceases to be purulent, the night-sweats disappear, and fever comes to an end. For other conditions, as chronic bronchitis, typhoid fever, and intestinal catarrhs, it should prove useful. Two instances of its use in pulmonary tuberculosis are briefly reported.—*Klinisch-Therapeutische Wochenschrift*, 1898, No. 19, S. 716.

Orexin as a Stomachic.—Dr. Frederick Kolbl has obtained good results from the use of this drug in chlorosis and anæmia, in anorexia of convalescents after severe illnesses, in nervous dyspepsia, hysteria, and neurasthenia. It may be given with good results in the diminishing appetite found in early and chronically developing pulmonary tuberculosis, in slight cases of gastric catarrh, in anorexia accompanying valvular dieases of the heart, and chronic myocarditis. In every case of vomiting in pregnancy this symptom was relieved in the course of a few days. It is contraindicated in all cases of acute inflammation and ulceration of the mucous membrane, and in all cases of hyperacidity and excessive gastric secretion, such as is found in acid dyspepsia. The dose is from one and one-half to three, increased to seven, grains daily, in a little cold water. The best time for the administration is about one hour before the chief meals—*e. g.*, dinner and supper—and it should not be followed by a large quantity of warm liquids. The best method is as a pill, each containing the minimum dose above given.—*The Therapist*, 1898, No. 5, p. 112.

The Action of Formaldehyde on Digestion.—M. LINOSSIER reports his observations made in the laboratory. The remarkable antiseptic properties of this substance and its relative harmlessness suggest its use as an intestinal antiseptic. Formal (a 40 per cent. solution of formaldehyde), in from 0.19 to 2.50 parts *per mille*, was employed. The results were that these solutions retarded all forms of digestion. This was most marked in the pancreatic digestion of albumins; the action of the saliva, gastric juice (pepsin and milk), and pancreatic amylase is not markedly hindered unless larger doses are employed than can safely be introduced into the alimentary canal.—*Bulletin Général de Thérapeutique*, 1898, 21e liv., p. 811.

Treatment of the Summer Diarrhœa of Infants.—Dr. ALEX. LEWERS believes that it is more rational to say that milk should be stopped because, under existing conditions, it has no chance of being digested than because it is a poison. As for irrigation, it probably has the same action internally as externally, viz., stimulant and sedative, the tone of the intestinal wall being improved and peristalsis lessened. As for routine treatment, if vomiting is not prominent, either castor-oil or calomel to clear the intestine, and then bismuth to soothe it. If vomiting is severe, small doses of calomel and Dover's powder, or stomach lavage, followed by bismuth, are still the most satisfactory. Astringents and antiseptics are useless, and there is no golden rule of diet except to stop the milk.—*Intercolonial Medical Journal of Australasia*, 1898, No. 4, p. 212.

A New Treatment of Syphilis.—M. LALANDE states that this medicament is obtained by the prolonged action of sodium chloride upon organic matter rich in keratin ($C_{51}N_{17}H_6S_5$). The keratin from the horns of ruminants is richer in sulphur. To prepare this the horns of a calf are powdered. The maceration, with daily agitation for a month, is conducted at a temperature of 77° to 86° F. The formula is as follows: Powdered horn, 60; sodium chloride, 10; distilled water. This is now left in a dark room for four months. After decantation it is heated for half an hour in a closed vessel at 194° F., and after cooling the vessel is opened. The liquid is limpid, clear, and yellow, of an odor suggesting burnt horn, and of a salty taste. It must be preserved from light. It contains gelatin, 5.30; calcium phosphate, 0.30; calcium sulphate, 0.03; sodium chloride, 8.37; potassium sulphate, traces, to water, 194° F., 1000.00. This has been employed in the treatment of thirty patients during the past two years: primary lesion, 9; secondary period, 19; tertiary period, 2. Of these ten had previously received mercury. The liquid is injected hypodermatically, under aseptic precautions, in dose of from one to three-fifths of a teaspoonful, into the infrascapular fossa or lumbar region, at from one to eight day intervals. The first injections are followed by pain. The liquid is rapidly absorbed. Three hours later appear a moderate elevation of temperature, some somnolence, and possibly sweating. There is no diuresis nor albuminuria; several times urea has been increased. Benefit generally follows the third injection; the mucous patches dry, the syphilides disappear, and ulcerations heal. From ten to thirty injections suffice for cure, which is not followed by relapses.—*Les Nouveaux Remèdes*, 1898, No. 8, p. 169.

The Treatment of Syphilis of the Nervous System.—Dr. J. W. Courtney insists upon the inadequacy of the usual two-years' treatment of the secondary manifestations as a prophylactic against the outbreak of this disease in the nervous system. Gowers recommends that every syphilitic subject, for at least five years after the date of his last secondary symptoms, should have a three-weeks' course of treatment twice every year, taking for that time twenty or thirty grains of iodide a day. If only one drug is to be used in the treatment of active cerebro-spinal syphilis, by all odds mercury by inunction is preferred, although the value of the combined use of this with iodide is unquestionably greater than that of either alone. The mercurial inunction should be vigorously prosecuted; a drachm should be rubbed in daily for six days, then allow an intermission of a day, on which the patient takes a warm bath. The process is then repeated (unless some decided contraindication arises) until thirty or forty inunctions have been made. In the mean time potassium iodide is ordered in increasing doses until a drachm or two are taken per diem. Attention to the hygiene of the mouth cannot be too strongly insisted upon during this time, and all tobacco and alcohol should be withdrawn. In treating symptoms due to the thrombotic occlusion of a vessel, it should be remembered that the thrombi are not syphilitic, and that consequently the patient's general condition may be seriously impaired by drenching him with iodide. To such gummata as do not yield to medicinal treatment the same rules for surgical procedure apply as for tumors of different nature.—*Annals of Gynecology and Pediatry*, 1898, No. 8, p. 574.

The Treatment of Gastric Hyperacidity.—Dr. Elliott P. Joslin states that if the motility is at fault the general condition is to be benefited, and rowing and golf, by strengthening the abdominal muscles, will give support to the overloaded organ. Massage by a skilful operator may empty the stomach, and so be of value. Electricity is of no use. Only one drug is of value, and that only when given in a particular way: ten drops of the tincture of nux vomica thrice daily, increasing one drop daily until a maximum daily amount of sixty to ninety drops is reached. Should these measures not suffice, the stomach-tube should be employed. This, however, is exhausting, and besides a large amount of food is in this way removed from the body. In one instance the estimation of the nitrogen in the stomach washings revealed the fact that nearly one-fourth of all the food given by the mouth was removed by the tube on the following morning. As the stomach absorbs no water, it is in these severe instances of hyperacidity associated with gastric dilatation that thirst is troublesome. This is best relieved by nutrient enemata of normal salt solution. Food is also absorbed extraordinarily well by the rectum, and in one instance 87 per cent. was so taken up. Rationally, an albuminous diet is wrong in hyperacidity, but all acknowledge the temporarily good effects of proteids, and in the milder cases the symptomatic treatment seems indicated rather than the rational: carbohydrates for the severe, and proteids for the mild form. Yet the author has almost invariable satisfaction with the proteid diet. On giving carbohydrates, since the ptyalin is soon destroyed in the stomach, foods in which the starch has been converted into dextrin are indicated or diastase administered. The pain arising from the acid itself may be neutralized in various ways, the

simplest of which is to dilute the acid with liquid. This, of course, should not be encouraged, for fear of producing trouble with motility, and thereby augmenting the existing condition. Another means is to give a diet which will combine with the excess; this is the albuminous. That this is powerful in its action is shown by the fact that two or three eggs will combine with a pint of 0.2 per cent. hydrochloric acid. Further, the products of proteid digestion—that is, peptones—have the power of combining with a greater quantity of hydrochloric acid than the original proteids. This fact suggests that it would be more rational to administer peptones. The aim should be to give food in small bulk, and such preparations as malted milk tablets may be of service. If held in the mouth for some time they may be useful in increasing the alkaline saliva; thus the organism furnishes its own alkali. As for the alkalies proper, all agree that they are temporarily of great use, though in time they lose some of their power and are purely symptomatic. Combined with bismuth, their action is promoted. The special variety is not of importance, but large doses must be given. No matter what the treatment or rule of diet is in any case of stomach trouble, it should be always borne in mind that the marasmus of probably all stomach patients, excepting those with cancer, arises from the diminished amount of food eaten. Patients can get along without a stomach, but not without food.—*Boston Medical and Surgical Journal*, 1898, No. 17, p. 389.

The Therapeutic Value of Strophanthin.—Dr. E. Stahr concludes that (1) the crystallized strophanthin from a particular maker given by the mouth is not a strong poison, in that it can be safely administered to five-eighths of a grain daily. (2) It has no cumulative action. (3) When given in quarter-grain doses it increases the flow of urine. (4) It has no untoward action. (5) The patients note that attacks of palpitation are not so frequent or severe. Whether this results from the drug or from the rest which they obtain in the hospital is not clear.—*Therapeutische Monatshefte*, 1898, Heft 5, S. 245.

[The large doses employed and the general results obtained from them are so entirely opposed to those obtained by competent observers that it is evident that the preparation employed was inert.—R. W. W.]

Fleiner's Method of Treating Hyperchlorhydria.—Dr. B. Olivette introduced through a stomach-tube from one hundred and fifty to three hundred grains of bismuth subnitrate suspended in water before breakfast each day for about three weeks. Four patients were under observation. From these he concludes that: (1) Large doses of bismuth are well borne by patients suffering from gastric ulcer and hyperchlorhydria, and give rise to improvement as to subjective symptoms not only during but after the treatment. (2) This improvement is not permanent, but more marked in ulcer than in true hyperchlorhydria. (3) The doses necessary for betterment vary from 150 to 230 grains for each administration. (4) The exhibition of these doses has no marked influence upon either the quantity of the gastric fluid or the motility of the stomach. (5) By this method the hydrochloric acid content in hyperchlorhydria was very slightly diminished, and this cannot explain the improvement in the subjective manifestations (gastric irrita-

tion, pain, and vomiting). The explanation appears to be that the bismuth forms a protective coating over the gastric mucous membrane which protects it from the hyperacid gastric juice.—*Therapeutische Monatshefte*, 1898, Heft 4, S. 181.

MEDICINE.

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New Bacilli Resembling those of Tuberculosis.—MOELLER, in a preliminary note (*Deutsche med. Wochenschrift*, 1898, No. 24), reports the finding of these bacilli on timothy and other grass and in the feces of cows, horses, swine, goats, and mules. (Tuberculin-tests showed the animals to be free from tuberculosis.) They resemble the bacillus of Koch in size and shape and in their resistance to acids and alcohol, but differ in rapidity of growth on various media and in various minor points described in the original. They also have certain points of difference as compared with the bacillus discovered in butter by Petri and Rabinowitsch, the bacillus of leprosy, and that of smegma. Severin and Capaldi have probably found the same bacilli in dung, but were not able to cultivate them, as Moeller was. The latter thinks there is a close relationship between the timothy bacillus and that of dung; and also, as the result of certain experiments described, that the various bacilli which resist acids may be made to assume the characteristics of others of the same kind. Inoculations with pure cultures gave interesting results. In guinea-pigs there was infiltration, with central caseation at the seat of inoculation. The lymph glands were enlarged; the pleural cavities contained an excess of fluid; the mesentery and peritoneum showed numerous small grayish-white and yellow nodules; similar nodules were in the spleen, which was much swollen, and the liver. The lungs contained nodules, and in one case cavities, one of which was three-fourths of a centimetre in diameter. The nodules and the contents of the cavity contained large numbers of the bacilli. Cats, rats, pigeons, and hens were immune. The changes, on the whole, were much like those of experimental tuberculosis, but the cavity formation was more like that in human lungs than anything usually seen in experiments. Histologically, the changes resembled those of tuberculosis to a certain extent. Giant-cells were rare, and, so far as examined, did not have peripheral nuclei. In some cases the lesions were not so much like those of tuberculosis. The further details will be awaited with interest.

Motor Tests and Disturbances of Motion in Sciatica and Lumbar Pain.—MINOR (*Deutsche med. Wochenschrift*, 1898, No. 24) summarizes as follows the results of some investigations he has made: 1. Patients with sciatica or lumbago are not able to rise from the sitting position with extended legs, without the use of the hands. In all cases of lumbar pain after cold, trauma, caries, etc., in which the pain is bilateral, the body is raised as in typical cases of pseudohypertrophy of the muscles; the arms are brought forward, and finally the body is extended.

2. In typical sciatica the arms are placed behind the body so as to displace the centre of gravity backward, then one hand is pushed from the floor, while the body is balanced on the other.

3. Inasmuch as these motor alterations are purely objective, they enable one to make a more accurate localization of the pain than can be obtained from the patient's statements, and also furnish more reliable guides to the intensity of the pain and its alteration during treatment.

4. In traumatism, as of the coccyx, these tests will show whether the injury is especially unilateral, even if the subjective sensation indicates affection of both sides.

5. Knowledge of these disturbances of motion, especially of that in sciatica, may be of great value in case of suspected simulation. So, if the patient uses the lumbar method in rising from the floor, simulation is probable.

The author advises that the method be studied at first in well-marked cases of lumbago and sciatica.

Toxæmic Delirium in Heart Disease.—EICHHORST calls attention to a condition sometimes seen in cases of cardiac insufficiency, especially in those cases in which rapid disappearance of œdema and cyanosis and greatly increased excretion of urine follow proper treatment. Eichhorst's routine treatment consists in the administration, when rest in bed alone is not followed by marked improvement, of powders containing 1 decigramme of digitalis powder, 1 gramme of diuretin, and 3 decigrammes of sugar. Three daily doses are given, and in most cases thirty powders are sufficient to restore compensation. The urine is always rapidly increased, and may amount to five or even seven litres daily. The symptoms to be described occur during the diuresis, and are more likely to appear in old persons than in younger ones. The first thing noticed is usually somnolence, which may be so deep that it is difficult to arouse the patient. Soon the consciousness is disturbed; the patients do not recognize their acquaintances or surroundings. Delirium then comes on, varying in degree up to violent mania. The respiration is often altered, being deep and frequent, without evidence of obstruction. The face is red. Muscular spasm was not observed in any case. These symptoms last until the œdema is gone, and polyuria has been followed by normal excretion, and then cease gradually or suddenly. In no case was there albuminuria; hence the author excludes ordinary uræmic poisoning, and attributes the symptoms to an intoxication from unknown bodies derived from the œdematous fluid and not excreted rapidly enough by the kidney. In no case was the condition actually dangerous.—*Deutsche med. Wochenschrift*, 1898, No. 25.

Metabolism in Acute and Chronic Leucæmia.—MAGNUS-LEVY (*Archiv für path. Anat., etc.*, Bd. clii., H. 1) gives the result of his examinations on the extensive material in the Urban (Berlin) Hospital. The differences in metabolism in the two classes of cases were very striking. In acute cases there was a large amount of urine and notable increase of total nitrogen and uric acid, with increasing excessive excretion of nitrogen up to the end. In chronic leucæmia there was a moderate amount of urine and normal nitrogen, without an excessive excretion before death. An excessive metabolism is characteristic of acute leucæmia, but, as the author shows from his own material and the literature, this is not constant. In contradiction to many other investigators, the author denies a constant relation between the number of leucocytes and the excretion of alloxur bodies, but finds these in some cases directly, in others inversely, proportional. On the other hand, he thinks that the constant loss of blood in acute leucæmia increases the albumin metabolism directly. He also thinks that the sudden diminution in oxidation injures the cells and leads to increased breakdown. One case showed an enormous increase of phosphoric acid, which the author thinks was derived from nuclein. In five cases of leucæmia he found uric acid four times; hypoxanthin was always present, sometimes in large amounts; xanthin only in traces; pleural and pericardial alloxur bodies in proportions similar to those in the blood, but the former contained less, corresponding to the smaller number of cells.

Pyopneumothorax Examined by X-Rays.—KIENBOCK (*Wiener klin. Wochenschrift*, 1898, No. 22) has made some interesting observations on a case of closed pyopneumothorax. The latter has existed for several months, on the left side. The heart and diaphragm, at first much displaced, gradually returned to their former positions, though the heart and mediastinum became adherent to the right pleura. The right lung showed signs of a tuberculous infiltration. The left lung was infiltrated and retracted. The fluorescent screen showed differences corresponding to the changes indicated by the physical signs, and not necessary to describe. The most interesting phenomena consisted of certain movements visible in the screen. In inspiration the level of the fluid rose about three-fourths the width of an intercostal space. The level of the fluid, in addition, showed waves independent of the respiratory changes, partly synchronous with the heart-beat, but there were also waves, seen when the breath was held, due to shaking of the floor. Percussion of the thorax also caused visible waves, and shaking the patient caused waves of considerable height, up to 10 cm. The author suggests several explanations for the respiratory change of the level of the fluid. It might be due to a contraction of the diaphragm, made convex downward by the fluid, or to increased abdominal pressure forcing the diaphragm up. Narrowing of the left side by displacement of the mediastinum was excluded by inspection with the screen. The pulsation of the fluid seems to confirm Traub's theory of the cause of pulsating empyema, viz., by direct transmission of the heart-beat to the fluid. That it was not due in this case to compression of air could be demonstrated by putting the patient in such a position that the heart was surrounded by air. The pulsation ceased, and only began again when the apex was submerged. The absence of visible or

palpable pulsation was, perhaps, due to the low tension of the air in the thorax, or to the thickening of the costal pleura, and makes it probable that pulsating empyema is much more frequent than ordinary methods of examination would lead us to think.

Severe Cerebral Symptoms and Death from Excessive Dilatation of the Stomach.—Von Jürgensen reports (*Deutsches Archiv für klin. Med.*, Bd. lx. p. 327) the case of a farmer, aged forty-three years, who for ten years had occasional attacks of pain, fulness, and sense of oppression in the epigastrium after meals. Anorexia and excessive appetite alternated; during the attacks vomiting was frequent. The patient was emaciated, pale, and sallow. The abdomen was distended, the superficial veins prominent. The stomach was much dilated in all directions, but the exact outlines were not made out during life. The patient refused mechanical treatment, and was treated by wet-packs, diet, rest in bed, and papain internally. The latter, the patient thought, lessened the tympanites, but the general condition became distinctly worse. The patient complained of great weakness and severe thirst. Water was vomited as fast as swallowed. The thirst was so great that in one night ten litres of water were drunk and vomited. Next morning the intellect was slightly clouded, there were atactic movements of the arms and legs, later general spasms. The symptoms grew worse. The urine, scanty before, was suppressed. The thirst and vomiting persisted. Coma came on, and irregular breathing, cyanosis, cold extremities, loss of reflexes, spasms, and vomiting continued until death ensued, two days after the change for the worse. Autopsy showed the greater curvature of the stomach at the level of the umbilicus; the lesser curvature formed an acute angle, the sides almost touching. The greater curvature measured 55 cm., the height from the fundus to the cardia 20 cm., the depth 14 cm. One centimetre below the pylorus was a stricture of the duodenum, caused by a ring-shaped scar. The duodenum above this and the pyloric part of the stomach were dilated; the duodenum between the stricture and the pyloric ring was the seat of a cicatrized ulcer, about 2 cm. in diameter, continuous with the constricting scar-tissue. The mucous membrane of the stomach was thin and smooth in the pyloric portion, unusually folded in the fundus, where there was slight *état mammelonné*, the two parts sharply separated by the fold of the lesser curvature. The muscular coat of the stomach was hypertrophic. The œsophagus was dilated and hypertrophic, the epithelium thickened in areas. The bladder contained 100 c.c. of urine, specific gravity 1020. There was a small amount of albumin, but no sugar. The other organs showed no alterations bearing on the case.

That the death was due to an intoxication of some kind was clear, and the author, showing that diabetes, cancer, and cholæmia were not present, in view of the enormous thirst and the failure of absorption, ascribes the fatal symptoms to lack of water in the blood and tissues and intoxication from the suppression of urine. It will be remembered that Kussmaul, in his earliest work on dilatation of the stomach, looked on lack of water from lessened absorption as of great importance, but afterward abandoned the idea. Von Jürgensen, however, is not willing to abandon the theory entirely, but would investigate each case in order to determine the precise

factors involved. He also suggests that stricture of the duodenum prevents the passage of the stomach-contents even more than obstruction of the pylorus.

Bolognini's Symptom in Measles.—KOPPEN has investigated this symptom, and not only has shown its worthlessness in diagnosis, but has also explained its cause. As described by Bolognini three years ago, the symptom is elicited as follows: The patient is placed on the back, the legs flexed. If then both hands are placed on the abdomen and the finger-tips used in palpation with increasing pressure, a feeling as of two rough surfaces rubbing on each other will be detected in circumscribed areas or over the whole anterior abdominal wall. With strong pressure or prolonged palpation the symptom disappears. Bolognini missed this only twice in two hundred cases. He found it in the earliest part of the prodromal stage, and it lasted until the end of the eruption. There were never symptoms of peritoneal irritation. The symptom was never found in healthy children, except among a few who had had measles a short time before. Koppen examined 316 cases of measles, some of them more than once, and with negative results in more than half. He found that the sensation was less like friction than like crepitation, as in emphysema of the skin, and that if the fingers were kept on the place when the sensation was discovered a coarser motion, evidently gurgling, would ultimately be felt and heard. In many cases, with the symptom there was diarrhœa, with foamy stools. On account of the partial absoption of the gas or water, or the expulsion of the flatus, the symptom does not always coincide with the condition of the stools.—*Centralblatt für inn. Med.*, 1898, No. 26.

SURGERY.

UNDER THE CHARGE OF

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A New Method of Tendinoplasty for the Dorsal Digital Tendons.—V. HACKER (*Wien. klin. Woch.*, 1898, No. 2) describes a new method which he employed in a case where there had been loss of the skin and of the extensor tendon of the index-finger, which was destroyed for a considerable distance, while the interphalangeal articulations were opened. The case came to him with a large granulating area covering the dorsum of the finger, and no sign of the tendon remaining.

The method employed was the utilization of the tendon of the index-finger in conjunction with the common extensor to bridge the gap. The extensor proprius indicis was divided at a point sufficiently near the carpus to enable him to reverse it and insert it beneath the periosteum and tendinous remains upon the dorsum of the last phalanx.

Its connection with the common extensor in the aponeurosis over the metacarpo-phalangeal joint insured its vitality, while it was united to it by a few stitches.

The skin defect was remedied by passing the finger beneath a bridge-shaped flap on the chest just below the xiphoid cartilage, holding it in that position till the flap had firmly united.

The result was all that could be desired, the function of extension was restored, and the skin flap united perfectly without adhesions to the tendon beneath.

The Treatment of Acute General Peritonitis Originating in the Vermiform Appendix.—DEANESLY (*British Medical Journal*, February 12, 1898) terms those cases general peritonitis where pus or septic fluid is found in the pelvic pouch, in both flanks, and among the small intestines as high as the navel, and when the inflammation is nowhere limited by firm adhesions. These cases demand immediate interference by operation to save the patient.

The author distinguishes three varieties according to their mode of origin: (1) The acute fulminating variety. There is an acute septic invasion, spreading with such rapidity that the greater part of the peritoneum is involved in twenty-four or thirty-six hours, and in which the virulence of the micro-organism is so great, or the resistance of the tissues so feeble, that the inflammation is not limited by the formation of durable adhesions among the surrounding intestines. (2) This variety follows the bursting of a localized abscess around the appendix by some mechanical cause, such as a blow or strain. (3) In this variety the onset is less acute and the early symptoms less severe. In fact, clinically, it appears to be an ordinary case of what used to be called typhlitis. There is little pain, moderate fever, a tender swelling in the right iliac region, and constipation, but no vomiting after the first day; but the swelling, instead of gradually disappearing, or coming to the surface in the form of a localized abscess, becomes lost in increasing general distention, which begins in the lower part of the abdomen and spreads upward.

In the treatment of these cases the author believes that strict antisepsis is as essential as if no pus was present, as a mixed infection increases the danger. He employs the median incision in order to reach readily all parts of the abdomen, using a second incision over the appendix if it is found necessary. In ligating the appendix he forms a peritoneal cuff, ligates close to the cæcum, and then ligates the cuff over this, tying off the mesentery separately.

The removal of the appendix he considers to be an essential part of the treatment of cases of general peritonitis. In case of localized abscess it is generally held, and he believes rightly, to be unwise to distuurb adhesions more than necessary, for fear of generalizing the infection. For the same reason, it is not advisable to prolong the search for the appendix after open-

ing and draining a localized abscess, and it is certain that leaving it in such cases is no bar to recovery. In general peritonitis, on the other hand, the only chance of averting a fatal result is the removal of the focus of infection and as much of the toxic products as possible. In other words, the appendix must be removed and all intestinal adhesions thoroughly separated in order to liberate and remove the collections of pus and lymph among the coils.

The abdomen he cleanses in all cases by means of sponges on holders, without the use of irrigation, which he has discarded.

Histological Study of the Varieties of Appendicitis.—Following the lines of his former work on this subject, PILLIET (*Le Prog. Méd.*, January 29, 1898) shows that the different forms of appendicitis may be classified according to the manner in which the follicles of that organ are affected by the process of inflammation.

Of calculous appendicitis he says:

1. The calculus is always composed of mucus and blood. Chemical analysis proves this and accords with the fact that mucus contains alkaline phosphates and carbonates. These calculi are therefore comparable to the calculi found in other organs of the body.

2. The appendicitis in itself is mild and prolonged. It is essential for the formation of calculi that the glands of Lieberkühn remain intact; that they are excited, but not destroyed. They can therefore coexist with recovery, though this form of appendicitis is, like all others, liable to acute exacerbations. It is generally, however, catarrhal in nature, in opposition to the acute ulcerating, perforating variety.

The so-called obliterant form of appendicitis is due to the destruction of the glandular tissues gradually and the formation of mucoid tissues in their place uniting the mucous membrane throughout, or it may be that the union takes place in the proximal portion only when its adhesion gives rise to the cystic variety. Gangrenous appendicitis is compared to the gangrenous form of dysentery, and is distinct from follicular lacerations.

Horseshoe Kidney Simulating Malignant Disease of the Abdomen.—OLIVER (*British Medical Journal*, February 26, 1898) reports an interesting case in which the symptoms exhibited by the patient pointed to malignant disease of the abdomen. A tumor was found in the median line that moved with respiration, produced pressure symptoms on the aorta, and seemed to have some connection with the symptoms of gastric disturbance displayed by the patient.

An exploratory incision showed a horseshoe-shaped kidney which rested upon the aorta and was slightly loosened from its median attachments.

The patient was relieved of all his troublesome symptoms by the operation. The author calls attention to the case as illustrating the fact that these abnormal kidneys must be taken into account in making a diagnosis in cases of tumors of the abdomen.

What Operation can do for Cancer of the Tongue.—In the study of one hundred and two cases of operation for cancer of the tongue in which he performed the primary operation, BUTLIN (*British Medical Journal*, Feb-

ruary 26, 1898) found that he had operated upon nearly an equal number in hospital and private practice; he also found, after getting returns from all but seven cases, that, besides the fact that the mortality was greater immediately subsequent to the operation in the hospital cases, the number of absolute cures with freedom from return, in some cases for thirteen years, and in all for over three years, was much greater in the private than in the hospital cases.

The number of deaths due to operation in the hospital group is no fewer than nine, while he only lost one of his private patients in whom there was marked involvement.

The number of such patients alive and free from disease more than three years after operation, or who died from other causes after the expiration of three years, was seven in the hospital group and thirteen in the other group, and there are nine others free from return after one year, but not over three years, while there are only two hospital patients who may prove cures. The percentage of cures in the hospital group of cases at the best is sixteen, while in the private cases it is twenty-six, with a chance of its being much greater.

The reason for this difference in results is found in the fact that the private patients are better educated and come for operation much earlier than do the others, which fact illustrates the great advantage of early operation.

The study of the cases which may be claimed as cured shows that the disease, in the large majority of them, was situated in the anterior two-thirds of the tongue. That is only what has been suspected and suggested, but hitherto he has not been able to prove it by a sufficient number of cases. But even some of the worst cases may be cured if the disease has not invaded the tonsillar and neighboring regions.

Among the private patients cured there was not one in whom the glands were removed, while in the hospital cases, of the seven five had the glands removed at the time of the operation or immediately subsequent to it by a second operation, and in four or five they were not only enlarged, but proved by microscopical examination to be cancerous. The author does not believe that the removal of the entire tongue is essential to a successful operation. The whole tongue was removed in only one of his successful cases. There is ample proof that removal of a portion of the tongue is sufficient to cure a considerable percentage of patients and to save a much larger percentage of cases from recurrence in the mouth. The removal of the entire tongue as a routine operation can be justified only by proving, as far as it is capable of proof, that a very considerable proportion of the persons who suffer from recurrence in the mouth would have been preserved from that recurrence in the mouth had the entire tongue been removed.

The author always aims at removing the cancer with three-quarters of an inch of apparently healthy tissue around it in every direction. Where the disease is on the border of the tongue his routine practice is to remove half the tongue to an inch behind the margin of the disease. In cases in which the disease is near the tip or forepart of the dorsum, the forepart of the tongue is removed.

The question as to the prevention of secondary infection of the glands of the neck is one of great moment, and yet its solution is full of uncer-

tainty. The author's tables show how frequently successful treatment of the cancer of the tongue has been spoiled by secondary affections of the glands which had appeared normal at the time of operation.

The expectant plan of treatment is full of uncertainty, as often the glands are so involved before it is possible to detect them that their removal is unavailing. On the other hand, it is difficult to say which group of glands will be involved, and consequently which should be immediately removed.

In spite of this uncertainty the author has become convinced of the necessity of a routine operation if cancer of the tongue is to be more successfully dealt with. He has employed during the past eighteen months the following operation, removing the entire contents of the anterior triangle of the neck: A very careful dissection of the triangle is made, so that the connective tissue and glands are all taken out in one continuous mass. Search is made between the muscles in front for one or two deeper-seated lymphatic glands, and the glands in front of the parotid gland and about the angle of the jaw are removed with the contents of the triangle. The submental and parotid glands are not so easily and certainly removed *en masse* in this operation as the submaxillary and carotid groups. This is done at a second operation, and not at the time of the excision of the tongue.

The Surgical Pathology and Treatment of Tuberculous Bone Disease.—NICHOLS (*Boston Medical and Surgical Journal*, January 27, 1898) finds that many observations prove that tubercular disease of the bones and joints is caused by the tubercle bacillus.

Injuries of moderate severity favor the production of the disease.

In bones the disease begins in the epiphysis, and is more extensive than appears on gross examinations. Hence, in operation for removal of the disease, a considerable margin of apparently healthy bone must be removed.

Tuberculosis of the joints is generally, if not always, secondary to tubercular disease in the epiphysis of adjacent bone.

Abscess formation is due to extension of the tubercular process to the soft parts. The contents and wall of the tubercular abscess are different from those of infectious abscesses. Partial removal of the abscess wall is harmful.

Repair is caused by the formation of fibrous tissue, which replaces and partly encapsulates the tubercular tissue. Repair may be incomplete. Fibrous tissue may produce fibrous anchylosis, or the tissue may become ossified and lead to bony anchylosis.

Paraplegia in Pott's disease is rarely due to direct bony pressure. Usually the pressure is caused by tubercular peripachymeningitis. Rarely the pressure causes degeneration of the cord.

In regard to the surgical treatment, CABOT (*Ibid.*) says that, accepting the proposition that in these cases of tubercular bone disease we are dealing only with the localized focus, and therefore the operation is necessarily incomplete, and that nature must even afterward do much to accomplish the cure, it is plain how important it is to the surgeon to do all in his power to improve the patient's general condition and so to arrange his operation as to obviate a long confinement in bed, if possible.

The removal of tuberculous material must be as thorough as it can possibly be made, the bony focus being always hunted up and entirely removed.

Where possible, a considerable surrounding portion of healthy bone should be removed with it. This is usually accomplished in resections of the knee and elbow, and also in tuberculous disease of the hip where the process is confined to the head of the femur. When, however, the pelvic bones are also implicated, this is more difficult to accomplish.

When tuberculosis attacks bones in their continuity, where it is impossible to remove considerable portions of the bone without seriously interfering with the function of the part, surgeons usually resort to curetting; this, as shown by the remarks of Dr. Nichols, should be even more extensive than has been supposed, and should remove apparently sound tissue.

In these cases recurrence has been very frequent, while in the carpus and tarsus it is almost certain to occur. This is due to their isolation and lack of nourishment. It is usually well, therefore, to wholly remove any of these bones that are diseased. In the wrist the functional result after a removal of many of the carpal bones is not good, but in the ankle these operations, when undertaken in the young, give excellent results.

The success of treatment in cases of tuberculosis, other things being equal, is in inverse ratio to the age; that is, the older the patient, the worse the prognosis. The extent of operation required in a particular instance is often a question which demands great judgment.

In the after-treatment hygiene and absolute rest to the part, with even, gentle pressure applied over the area, the wound being properly drained, are the essentials. The author believes that iodoform has, in a measure, a specific effect upon tuberculous material. It seems to act by stimulating the tissues to an adequate resistance to the tuberculous material left after operation.

In speaking of the conservative treatment of tuberculous joint disease, BRACKETT (*Ibid.*), says: the conservative treatment depends upon the cicatrization in the tubercular process, which at some stages of the disease walls off the affected portion.

The object of the treatment is to protect the diseased joint surfaces from all such irritation as may be in its respective stages injurious until the process of repair is complete. These indications, which are met with in a diseased joint, must be treated in a most thorough manner, demanding a continuance of the utmost care for a long period of time, and may be considered under the heads of immobilization, avoidance of jar, traction, and the removal of weight. The immobilization must provide against both voluntary and involuntary movements; this is done by the use of the frame. Traction must be sufficient to overcome spasm and carried out in the line of the deformity, and must be continuous both when it is applied by weight during the recumbent and by the apparatus during the ambulatory treatment.

The necessity for the removal of pressure by weight-bearing lasts longer than either that of immobilization or traction. During the ambulatory treatment this is accomplished either by the use of crutches while the traction splint is worn, or, later, by the apparatus used as a walking splint, as an ischiatic crutch, transferring the weight to the tuberosity of the ischium.

This method of treatment is most successful in the early stages of the disease, though cases can be cured by persistent effort in the later stages.

This treatment has the advantage of procuring results with the least destruction of bone, and the possibilty of a joint mobility is greater.

Splenectomy for Rupture Without External Wound.—An interesting and rare case of splenectomy is reported by BALLANCE (*The Practitioner*, April, 1898), in which the patient, a boy, fell from a tree a distance of ten feet, striking upon the abdomen on a pile of paving-stones. When admitted he was a little pale and collapsed, and obviously suffering much pain. There was great pain on palpation and percussion of the left hypochondriac region, but no pain or tenderness was found in any other part of the abdomen. There was no bruising of the skin or fracture of the lower ribs. A catheter was passed and urine and blood withdrawn, the latter being intimately mixed with the urine. There were also fractures of both forearms. The temperature was 97.6 and the pulse 70.

The following day the pulse-rate rose to 130, and the temperature to 100.8°. There was present great restlessness, respiration thoracic, shallow, and quick, alæ nasi moving. Abdomen slightly distended, immobile. No resonance anywhere. There was a large area of fixed dulness in the splenic and left lumbar regions.

Immediate operation was determined upon. The abdomen was found full of clots, the spleen ruptured. The spleen was removed, and the abdomen sponged out and closed. The patient made an uneventful convalescence.

For two days the temperature was 101° and the pulse over 100, but afterward both pulse and temperature were normal. For restlessness and sleeplessness morphine was given. During convalescence no serious symptoms supervened such as occur after the removal of the normal spleen in adults. About three weeks after the operation the lymphatic glands on the right side of neck, in both axillæ, and in the left groin were found to be obviously enlarged.

The symptoms produced by excision of the spleen in adults are: Progressive loss of strength and loss of weight. Extreme anæmia; aspect withered and sallow. A daily rise of temperature. Increased frequency of pulse. Fainting attacks, with great pallor of surface. Headache, drowsiness, thirst, voracity. Diminished or increased secretion of urine. Griping pains in abdomen and tenderness along the long bones. Enlargement of lymphatic glands. Certain typical blood changes. These symptoms become manifest some ten or fourteen days after operation, continue in great severity for a fortnight, and then a slow but perfect convalescence ensues.

The diagnosis of ruptured spleen is arrived at from the locality of the injury, the evidence of internal hemorrhage, and the large fixed dulness in the left flank.

The fixed dulness in the left loin and splenic region is not present in intra-abdominal hemorrhage from other organs, and is caused by the region being occupied by large quantities of clot. The dulness, therefore, cannot change with position, and is pathognomonic of this injury. The shock is at first very severe, and is due to the blow on the belly and the sudden escape of fluid into the abdominal cavity, as well as the great loss of blood. However great the initial collapse in uncomplicated splenic ruptures may be, experience warrants the belief that the bleeding will, at any rate for a time,

be arrested by the formation of clot in the ruptures and around the organ, and that in this way a rally will take place sufficient to justify the hope of successful surgical interference. The rhythmic undulations of size to which the spleen is subject possibly interfere with the continuity of hemorrhage from the organ and facilitate the formation of coagulum around it.

In the treatment the author advises excision. In his judgment any other plan is, even if feasible, dangerous. A patient with a belly full of blood is in desperate straits; the immediate danger to life is great, the operation must be completed in the shortest possible time, and no method less radical than excision can unequivocally be relied upon to arrest the hemorrhage.

In the treatment of the symptoms that arise after excision various remedies may be used, as fresh sheep's spleens lightly grilled, or extract of fresh spleen made with normal saline solution, red bone-marrow mixed with anchovy paste to make it palatable, arsenic, and cod-liver oil.

DISEASES OF THE LARYNX AND CONTIGUOUS STRUCTURES.

UNDER THE CHARGE OF

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OF PHILADELPHIA.

Amaurosis Following Intranasal Operation.—Dr. Francis R. Packard, of Philadelphia, reports (*Medical News*, October 9, 1897) a case of temporary amaurosis of twenty to thirty minutes' duration, occurring suddenly in a man, aged thirty-six years, the day following the removal with the cold-wire snare, under cocaine anæsthesia, of a small piece of hypertrophied tissue from the anterior extremity of one of the middle turbinated bodies; and he refers to a number of similar cases reported by Ziem, Bresgen, and Thorner. In some of these instances examination of the optic field was negative, but in a number of them congestion of the optic papillæ with venous pulsation was noted. Packard calls attention to the anatomical relation between the venous, arterial, and nerve connection of the eyes and nasal passages. Incidentally he alludes to a number of other sequelæ of intranasal operations, some of them quite serious. [It is well for these examples to be occasionally laid before the profession and reproduced from time to time.]

Primary Lupus of the Larynx.—Dr. Emil Mayer, of New York, reports and depicts (*New York Medical Journal*, January 1, 1898) a case in a Russian man, aged thirty years. To this he adds a second case reported by Dr. Morris J. Asch in 1881, and still living in 1897, with a knotted stub of epiglottis, though death occurred before the end of that year. These cases are followed by a summary of other cases and the opinions of their reporters, with some general remarks on diagnosis, prognosis, and treatment, and a bibliography of a series from which the study has been made.

Intubation of the Larynx to Control Vomiting.—At the recent meeting of the British Medical Association (*British Medical Journal*, October 16, 1897) DR. CHARLES LYMAN GREENE, of St. Paul, Minn., read a paper upon "The Feasibility of Controlling Pernicious Vomiting by Means of Intubation of the Larynx with a Specially Adapted Tube." The tube is, in general, like the O'Dwyer tube, but differs in certain particulars. The speculation is a theoretical one, seemingly supported by experiments upon the lower animals, and apparently confirmed by a case recently under Dr. Schadle's treatment for chronic laryngeal stenosis by forcible dilatation. This patient was wearing a tracheotomy tube, and, during the passage of the large œsophageal bougie employed, the man makes the most violent efforts to vomit, but cannot expel any of the contents of the stomach, whether that organ be full of food or not.

Death after Thyroidectomy.—MR. F. T. PAUL, of the Royal Infirmary, London, reports (*British Medical Journal*, 1898, No. 1931) a fatal case of thyroidectomy, the only one in some thirty-six operations. It was one in which symptoms of Graves' disease accompanied a rather large parenchymatous goitre in a girl, aged fifteen years, who died two and one-half days after the operation.

A second case is mentioned in which severe symptoms occurred after operation on a woman, aged forty-three years, with rather chronic Graves' disease; but, fortunately, she recovered.

Foreign Body in the Nose for More than Forty Years.—The *Journal of the American Medical Association*, 1898, No. 24, reproduces from the French Congress of Otology and Laryngology an item in reference to a patient complaining of purulent rhinitis, in whom a canula was found in the left nasal passage into which it had been inserted forty-two years before and forgotten. A radiograph showed that the upper end of the canula was still in the nose. It was extracted through the inferior meatus, when all disturbance ceased at once.

Foreign Bodies in the Bronchi.—An exceedingly instructive case is narrated by DR. ALFRED AUSTIN LENDON, of Australia (*Intercolonial Medical Journal of Australasia*, 1898, No. 3), and forms a prelude to an admirable little article on the subject.

A boy, seven years of age, while taking a bath January 28, 1888, accidentally inhaled an ebonized stud, and for three days felt pain in the chest at a spot corresponding with the second right costo-sternal articulation. Despite this history, he was treated for more than a year for asthma, the story of the foreign body being discredited. In January, 1889, he had an attack of acute pneumonia of the right lung, commencing with a moderate hæmoptysis, and on the subsidence of the attack his expectoration became purulent, very offensive, and more profuse than it had been before.

March 2, 1889, he was admitted in the Children's Hospital, under the care of Dr. Lendon, with great dyspnœa, frequent short hollow cough, and profuse fœtid purulent expectoration, amounting to a half-pint in the twenty-four hours. Exploratory punctures with the aspiratory needle securing pus,

an inch or more of the fourth rib at the right upper axillary region was excised, and the pleural cavity opened, but there was no pus; the lung itself was then punctured, and still no pus could be reached. Some slight recent adhesions between the upper and middle lobes were then broken down with the finger, and this liberated a small quantity of pus. The finger reached the pericardial sac in the nipple-line, and the lung was felt to be retracted from the clavicle. While manipulating, Dr. Lendon felt what he believed to be the stud, with some lung tissue intervening between it and the finger, but subsequent digital exploration failed to detect it again. A drainage-tube was inserted and the wound sewed up. No sooner had the doctor left the hospital than the boy expectorated the stud in a violent paroxysm of coughing, during which he said he felt it in its old place in his chest. For a few days he had great dyspnœa and rapid pulse, but soon recovered, and has been in perfect health for several years, the right chest showing but little indication of the former trouble there.

Intolerance of Metallic Tracheotomy Tubes.—At a recent meeting of the Ohio State Medical Society (*Journal of the American Medical Association*, 1898, No. 21) DR. ALBERT RUFUS BAKER, of Cleveland, reported a case in which a patient, tracheotomized on account of dyspnœa due to paralysis of the posterior crico-arytenoid muscles, found great difficulty with metallic tubes, which would break, get out of repair, and be hard to clean, and frequently cause excessive granulations around the orifice. The metal tube was replaced by a soft rubber one, which was more satisfactory.

Fatal Dyspnœa from Hypertrophied Thymus Gland.—I. Under the title of "A Case of Spasmodic Dyspnœa," DR. J. S. BARNETT reports (*Lancet*, 1898, No. 3896) a case of labored breathing, with suprasternal and subcostal recession, locomotion of the larynx, and dilatation of the nares during respiration, which developed in a male child soon after birth. Tracheotomy was performed, and the child improved for several weeks, when he died during a prolonged attack of dyspnœa. The autopsy showed the trachea and larynx to be normal, and the thymus gland enlarged, the outlying lobules of the latter extending well into the root of the neck. It is thought possible that this may have caused pressure upon the recurrent laryngeal nerves, giving rise to spasm of the larynx, which was relieved by tracheotomy, the fatal termination being caused by further enlargement of the gland.

II. In the *Münchener med. Wochenschrift*, 1898, No. 11, DR. O. CLESSIN reports a sudden death due to a large thymus gland. A healthy child, two months old, was found dead in bed one morning, though it had seemed well at ten o'clock the evening before, and had never suffered from cough or dyspnœa. On post-mortem examination the lungs were found congested and showed petechial hemorrhages, with signs of slight bronchitis. There were petechiæ in the heart muscle also. All the other organs were normal, except the thymus gland, which was so much enlarged as to overlie about two-thirds of the heart, while it furthermore compressed the trachea to such an extent that a pin could scarcely be passed into the tube. The thymus was dark, full of hemorrhages, and contained a large amount of dark secre-

tion. The sudden death was attributed to acute swelling of the thymus, but the manner in which this arose was not clear. Friedleben was unable to cause such death by killing pups by strangulation, and Clessin has failed in attempts to produce it by tying the thymus veins.

Hemorrhage from the Internal Carotid Artery in Sequence to Tonsillitis. I. Hemorrhage through the Ear.—WALTER H. BROWN reports (*Lancet*, No. 3901, abstracted in the *Philadelphia Medical Journal*, 1898, No. 26) an instance of profuse hemorrhage from the right ear in a child five years of age recovering from an attack of follicular tonsillitis, the source of hemorrhage eluding examination. The meatus was packed with iodoform gauze, and four days later another hemorrhage, more copious than the first one, took place four hours after the removal of the packing. Under the belief that the hemorrhage was due to an erosion of the walls of the internal carotid artery, in probable sequence to some inflammatory change surrounding the vessel, it was deemed advisable to ligate the common carotid artery at once. This was done, and the child made a slow but satisfactory recovery.

Croupous Tonsillitis of Staphylococcus Origin, with Fatal Perforation of Internal Carotid Artery. II. Hemorrhage through the Nose and Mouth.—Under the title of "Septic Perforation of the Right Internal Carotid Artery, DRS. A. JACOBI and JAMES EWING, of New York, report (*Philadelphia Medical Journal*, 1898, No. 23) a case of croupous tonsillitis of staphylococcus origin with fatal perforation of internal carotid artery in a child whose age and sex are not mentioned, and whose clinical history was not learned until five months after death.

The child took ill two weeks before death with ordinary symptoms of croupous tonsillitis, with membranes on both tonsils. There were no Klebs-Loeffler bacilli. In a week the throat was pronounced clear, and the child much better. Then came on a severe chill, with rise of temperature, pain in the throat, dysphagia, and swelling of the lymph-nodes on both sides of the neck. Two days later there was considerable hemorrhage from the nares and pharynx, and two days after a second very profuse hemorrhage, "filling a bowl" with apparently arterial blood. Two days later a third hemorrhage. The nares were then plugged. Two days later the plugging was removed from the nares while the child was struggling, and a fourth severe and fatal hemorrhage occurred from the nares and the mouth. The autopsy revealed the fatal hemorrhage from an ulcerous perforation of the right internal carotid artery and the pharyngeal wall, while the condition of the internal jugular vein indicated that it or its branches were possibly the origin of the first hemorrhage.

The account of this case is followed by references to some similar cases of pharyngeal hemorrhage.

Pneumococci in the Throats of Healthy Persons.—At a meeting of the Société des Hôpitaux of Paris (*Philadelphia Medical Journal*, 1898, No. 21) BEZANCON and GRIFFON reported the result in their studies relative to the presence of pneumococci in the throats of healthy persons, having employed the serum of a young rabbit as a culture medium. They examined, bacteri-

ologically, the secretion from the tonsils of forty persons of all ages, living under the most diverse conditions, and found pneumococci in every individual. They believe that imperfect methods of investigation must have been pursued in previous observations in which pneumococci were found in a much lower proportion of healthy throats.

Chronic Nasal Catarrh.—The Chicago Medical Society is to be congratulated at having had read before it (*Journal of the American Medical Association*, 1898, No. 21), by DR. H. GRADLE, an admirably sensible paper upon "Classification of the Lesions Constituting the so-called Chronic Nasal Catarrh," the details of which would make too great a demand on our own columns, and so we cordially recommend the original to our readers.

Anomaly of Tongue and Palate.—W. M. HELSHAM (*Australasian Medical Gazette*, April 20, 1898; *Philadelphia Medical Journal*, 1898, No. 26) reports a case of absence of the soft palate, with a cleft tongue, in a child otherwise normal. The tongue was completely divided from behind to the extreme tip, where the two parts were united.

Gunshot-wound of the Pharynx.—DR. EMIL ARONSON, of Dallas, Texas, reports (*Journal of the American Medical Association*, 1898, No. 21) a case of injury of the pharyngeal cavity from the explosion of a gun. The upper incisors were struck first and knocked out. The projectile then changed its direction and passed through the oral cavity to become embedded in the muscles of the pharynx, where it lodged on one of the vertebræ, but nevertheless the patient for twelve hours did not believe that there was a foreign body in his throat. The bolt was removed with the aid of forceps and retractor, and was four and one-half centimetres long and weighed nineteen grammes.

Fatal Secondary Hemorrhage after Removal of Adenoid Vegetations.—DR. WALLACE PREBLE reports (*Boston Medical and Surgical Journal*, May 19, 1898; *Philadelphia Medical Journal*, 1898, No. 22) a case of fatal secondary hemorrhage on the eighth day after operation.

Leptothrica Mycosis of the Pharynx.—DR. MAX TOEPLITZ reports (*New York Medical Journal*, 1898, No. 1021) three cases of the disease out of dozens which he has observed during the last twelve years, and presents a comprehensive summary of the subject, followed by a copious bibliographical record. His own cases were cured by the sharp spoon and the electric cautery.

Urticaria of the Pharynx and of the Larynx.—I. In the *Philadelphia Medical Journal*, 1898, vol. i. No. 14, DR. JOHN MADISON TAYLOR, of Philadelphia, reports a "Case of Urticaria of the Pharynx Producing Grave Œdema of Glottis." A lady, on attempting to sing shortly after dining, found herself unable to do so, without any well-defined reason. A forcible attempt to sing produced a sense of discomfort confined chiefly to the throat, and this finally grew so great as to produce exhaustion, apparently due to impending suffocation. When seen by the doctor shortly afterward, the face was livid

and cyanotic, the head thrown back, with gasping respiration and fluttering pulse, but without any mental disturbance. In some ten minutes or so urticaria appeared upon the skin and spread rapidly over almost the entire body, continuing for several hours, slowly subsiding in the next few days, and then occasionally recurring. The distress in respiration gradually diminished with the subsidence of the urticaria, and in a few days the patient was fully restored. The patient had been subject to attacks of severe urticaria twice before, but without complication on the part of the larynx.

II. Dr. Frank Woodbury recently read a paper (*Philadelphia Medical Journal*, 1898, vol. i. No. 20) on "Urticaria of the Larynx Causing Asphyxia in an Adult." A merchant, subject to transient tumefaction of the face, had an attack after a mid-day lunch of bread, cheese, mustard, and beer and whiskey. In a short time he gasped, turned black in the face, and fell on the floor, where he remained for a few minutes apparently dead. Respiration being restored artificially, it was found that urticarial lesions were present upon the thigh and back. The patient seemed perfectly well again after a couple of hours' rest in bed.

[It is quite probable that the dyspnœa in this case was due to œdema of the larynx, as was inferred by Dr. Taylor in his own case reported above.]

Changes in the Larynx and in the Trachea in Cases of Leucæmia.—The *Philadelphia Medical Journal*, 1898, No. 23, summarizes from the *Münchener medicinische Wochenschrift*, April 19th, a report by Otto Barnick, of a case of a boy, aged thirteen years, who was the subject of leucæmia. He had attacks of severe dyspnœa, with croupy cough. Laryngoscopic inspection revealed great thickening of the ventricular bands, and infiltration of the whole upper portion of the larynx. The child died with severe dyspnœa, aphonia, and bleeding from the mouth and nose. On post-mortem examination it was found that the infiltration of the larynx and bands was due to a dense collection of lymphocytes in the submucous tissue. The capillaries, also, were distended with lymphocytes, and these cells were especially abundant in the interglandular spaces. The submucous tissue in the trachea was affected in the same way.

Dr. Otto Barnick, clinical assistant in Prof. Habermann's Clinic at Gratz, contributes an elaborate article to the *Münch. med. Woch.*, 1898, No. 19. He reports three new cases in the clinical services of Professors Escherich and Kraus, the pathologico-anatomical material being contributed by Professor Eppinger, the best authority upon this subject. The lesions, as pointed out by Virchow, are lymphoid nodules, assimilating tubercles in different portions of the respiratory mucous membrane, with infiltration, which is sometimes so great as to require tracheotomy, which, in its turn, often merely postpones the fatal termination.

[This condition is unknown to most practitioners, and this article is therefore highly commended to those engaged in the study and practice of laryngology.—Ed.]

OBSTETRICS.

UNDER THE CHARGE OF

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Symphysiotomy.—LEPAGE (*Annales de Gynécologie*, March, 1898) reports eight symphysiotomies for contracted pelves. In all of these cases the degree of contraction was not a pronounced one, and the cases came within the recognized limit of the operation.

In his first case the child was lost through birth-pressure, the mother making a good recovery. The patient had broken her right thigh when a child, and the pelvis had suffered in consequence. The child perished from pressure upon the cord and pressure during extraction.

His second patient had lost two children by forceps extraction. In the present labor the symphysis was easily opened, the head rotated by Farabeuf's lever, and delivery was spontaneous.

His third case was a face presentation in a woman who had been pregnant eight times, and had lost several children by the use of forceps. After symphysiotomy the chin rotated under the pubes and the child was expelled.

In Case 4 fruitless applications had already been made with forceps before the patient came to the hospital. After symphysiotomy, the head was rotated by the lever and a living child delivered. The mother suffered from incontinence of urine for some time, and was threatened with phlebitis.

In Case 5 the mother had intestinal disorder and pain in one leg after the operation, but mother and child made a good recovery.

In Case 6 the forceps and lever failed to extract the child, and symphysiotomy was finally successful. The child, however, perished. A fracture of the skull was found upon autopsy.

The seventh operation was done upon a woman on whom symphysiotomy had been performed in previous labor. Some difficulty was found in opening the scar-tissue left by the first operation.

The eighth operation was done upon a patient who had a normal pelvis but a very large child. Both made a good recovery.

These cases illustrate fairly the legitimate field of symphysiotomy, and are of interest as clinical records.

In the same journal, April, 1898, PINARD reports seven symphysiotomies during the past year. There were 97 patients during this time in whom abnormality of the pelvis was recognized. Of these 77 had spontaneous births. Craniotomy was done upon 6, and abdominal section upon 3; the forceps was used three times, and symphysiotomy was done upon 7. These cases resemble the preceding. The forceps was used in tentative traction, and this failing, the symphysis was opened. Version was employed in some

cases. In several lacerations of the vaginal wall occurred, which were immediately closed by catgut stitches. Six of the patients had rhachitic pelves; one an obliquely contracted pelvis. Two were primiparæ, five multiparæ, and one of these had the operation the second time. In three labor was ended by forceps, in four by version. Six women and all the children recovered. One woman died.

The patient who died was a primipara who had albuminuria. The wound was found united apparently, but on further study was discovered to be infected. In the peritoneum were found pure cultures of the colon bacillus. In the symphysis were streptococci, staphylococci, and colon bacilli, and in the uterus the same germs were present.

The Use of the Curette in Subinvolution During the Puerperal State.—In the *Archiv f. Gynäkologie*, 1898, Bd. 55, Heft ii., KNAPP reports his experience in cases of subinvolution in which he has curetted the uterus during the puerperal state.

His patients were twelve in number. None of them had fever after childbirth, and there was no adequate cause for subinvolution which could be detected. All ordinary methods of securing involution had been faithfully tried. As indications for the operation, the following were considered sufficient: First, the retention of placenta or membranes within the uterus; second, the failure of other methods to secure involution; third, when the patient could not wait for prolonged involution to occur. Many working women could not afford to remain in the hospital a sufficient length of time without operation.

It is especially interesting to note that no adequate cause was found in all these cases for failure of involution. The exact manner in which the placenta was removed seemed to make no difference. Neither rapid nor slow labor seemed to influence involution. It was as common after premature birth as after labor at term.

The general health of the patient, however, had a marked influence upon involution. If the patient was strong and well, subinvolution rarely occurred. In primiparæ involution took a longer time, but was more complete when finished. In multiparæ it was not so perfect. Fever, excessive distention of the uterus during pregnancy, and all obstetric operations seemed to favor subinvolution. Repeated examinations during labor had the same effect. Especially important in securing good involution was the emptying of the bladder and rectum during the puerperal period. Where these were allowed to become distended, the uterus failed to contract. In cases where placenta or membranes were retained, the blunt curette was employed, but where hypertrophied decidua was present a sharp curette was used. The operation was based upon the size of the uterus, the condition of the cervix, and the condition of the lochial discharge. When the womb was large and soft, when the cervix was either partly open or tightly contracted and the lochial discharge was not red or serous, the case was considered a proper one for curetting. The occurrence of bloody lochia alone was not thought a sufficient cause. Knapp is accustomed to examine his patients ten days after labor, and if involution is not properly proceeding, curetting is performed. He is accustomed first to introduce a sound to determine the

size of the uterus, and observed that this is sometimes followed by contraction of the womb. He calls attention to the fact that the womb may be high in the abdomen and still involution have gone on well.

His cases all improved rapidly and were discharged cured six days after the curetting. Ergotin was given after the operation with good results.

Ectopic Gestation.—In the *Boston Medical and Surgical Journal*, May 12, 1898, SWIFT reports two interesting cases of ectopic gestation. In the first, a young woman recently married had symptoms of ectopic gestation, but no tumor could be found on vaginal examination. Membrane was discharged from the uterus, which Dr. Whitney pronounced characteristic decidua. On abdominal section, the right tube was found behind the uterus in such a position that it could not have been felt by vaginal examination. It contained an early embryo. The second case was that of a primipara who had an abdominal gestation with pseudo-labor and death of the child. Six weeks afterward Swift removed the fœtus and appendages. The patient made a good recovery. Whitney adds to this clinical report an interesting description of the decidual cells found in the first case and of the fœtus removed in the second.

Successful Antiseptic Precautions.—In the *Wiener klinische Wochenschrift*, No. 18, 1898, MARS describes the antiseptic precautions which he employs in Krakau. His results are excellent, a mortality of 0.4 of 1 per cent. for all cases in 700 patients, and a septic mortality of 0.28 of 1 per cent. His methods are practically to disturb the patient as little as possible. No douches before or after labor are given. The thighs and external parts are thoroughly scrubbed with soap and water and lysol. The hands of those who make examinations are very carefully cleaned, and the hand is always wet with antiseptics when the examination is made. Lacerations are closed immediately after labor. All operations or interference are done under strictly antiseptic precautions.

Cæsarean Section, with Transverse Incision of the Fundus.—SCHRÖDER (*Monatsschrift für Geburtshülfe und Gynäkologie*, 1898, Band vii., Heft 2) reports the case of a primipara with contracted flat pelvis, at full-term pregnancy. The patient elected Cæsarean section, with prevention of further conception. At operation the womb was opened by transverse incision of the fundus. The broad ligaments were ligated, the womb amputated, and stump covered with peritoneum and dropped. Bleeding was detected near the left sacro-iliac joint, with formation of retro-peritoneal hæmatoma. The peritoneum was incised, the clot turned out, and the vessel tied; bleeding promptly ceased. During recovery there was slight fever, tenderness over the stump, and infiltration of the tissues about the stump. This gradually disappeared, and recovery followed.

His next operation was for osteomalacia, and was a typical cœlio-hysterectomy, with intrapelvic treatment of the stump; recovery was delayed by a mild catarrhal pneumonia; the patient ultimately regained good health.

Case third was a rhachitic imbecile, with flat, rhachitic pelvis. This patient had been long in labor, and had been examined by a number of doctors and midwives. The child was dead, and the amniotic liquid dis-

colored. Cœlio-hysterectomy was performed. Septic peritonitis developed, and the patient died. She had evidently been infected before operation.

Case four was a patient with flat, rhachitic pelvis, in whom the child was delivered by the transverse incision of the fundus. The uterus was closed by suture, and good recovery followed.

[In reviewing this interesting series of cases the formation of retro-peritoneal hæmatoma in the first is of interest. In the reviewer's experience, a similar complication arose after Cæsarean section, which followed injury to the vessels of the broad ligament by a clamp. The hæmatoma in this case was large, was limited by peritonitis, and disappeared after incising the posterior vaginal vault, washing out the cavity with salt solution, and packing with gauze. In this case the hæmatoma developed several days after operation, and with no other sign of bleeding.

In case third the strong probability of previous septic infection would, in our experience, have suggested embryotomy rather than Cæsarean operation.—ED.]

In the *Centralblatt für Gynäkologie*, 1898, No. 24, HEIDENHAIN reports two successful Cæsarean deliveries in patients having pronounced osteomalacia. In both the fundus was incised transversely, the incision afterward closed with catgut. In both patients the tubes and ovaries were removed, but the womb was left. The results were good, the osteomalacic pains growing less and the general condition improving.

From the Lemberg Clinic, SOLOWIJ reports (*Centralblatt für Gynäkologie*, 1898, No. 25) a Cæsarean delivery for osteomalacia in which the uterus was left and the tubes and ovaries removed.

[It is difficult to understand the reason for leaving the uterus after the tubes and ovaries are removed. The weight of modern opinion is distinctly against such a proceeding.—ED.]

Rubber Gloves in Obstetric Work.—DÖDERLEIN (*Centralblatt für Gynäkologie*, 1898, No. 26) has for some time caused rubber gloves to be used by all who make vaginal examinations during labor. The results have been most satisfactory: lower puerperal temperature, shorter recovery, and less and cleaner lochia. The advantages of the use of gloves are (1) they are impermeable; (2) they are easily sterilized in steam, boiling-water, or antiseptic solutions; (3) when wet in 1 per cent. lysol they are smoother than the hand; (4) they are not so expensive that they cannot be used. The gloves are boiled for half an hour and then placed in 1 per cent. lysol. The hands are first sterilized as carefully as possible, the gloves are filled with 1 per cent lysol, and a glove is put on the hand used in examinations.

Does the Uterus in the Normal Puerperal State Contain Germs?—This much-discussed question is answered by BURCKHARDT (*Centralblatt für Gynäkologie*, 1898, No. 26), from the results of experiments in the Basil Clinic. He found that the uterus after labor is essentially an open wound, and that, like all open wounds, it is at first germ-free, and then contains bacteria.

In the puerperal state, at the eleventh or twelfth day, the uterine lochia is no longer sterile, and any unusual exertion which may alter the position of the uterus and interfere with its drainage is followed by slight absorption

and rise of temperature. In the greater number of cases bacteria gain access to the uterus through the cervix during the puerperal state, but they are not virulent germs, and if the uterus has not been wounded they occasion no trouble.

PÆDIATRICS.

UNDER THE CHARGE OF

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ASSISTED BY

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An Anomalous Case of Infantile Scurvy.—G. S. DE GROUTE (*Medical Record* April, 9, 1898) records a case of what seems by the therapeutic test to have been infantile scurvy, but presenting scarcely any of the classical symptoms of the disease. The child was a female, nine and one-half months of age, of good family history, and under good hygienic surroundings, never previously ill, and a picture of health. The first symptoms were pain in the right knee and hip, noticed only when certain movements were made, as when the nurse changed the napkin or made extension of the leg; neither joint was tender to pressure. Within twelve hours the pain was manifest also in the left shoulder and over the ribs, and soon after in the other limbs and the pelvis, though worse in the left arm and leg. There was only a slight swelling of the knee-joints; but no fusiform swelling of the lower extremities, no ecchymoses until late in the course of the disease, no changes in the gums.

The details of feeding were: Breast nursing for ten days; modified milk [no details stated] up to four and one-half months; then milk, barley-water, and water, of each two ounces; lime-water, one teaspoonful, sweetened to taste with milk-sugar and pasteurized. This last mixture was made stronger by increasing the milk and decreasing water until half milk and half barley-water; then the barley-water was made thicker. For a month before symptoms were noticed the child was taking at a feeding six ounces of this last mixture. As soon as the diagnosis was made pasteurization was stopped, and the juice of half an orange at first and later of a whole orange was given. Forty-eight hours after beginning orange-juice there was marked improvement, and in four days the child was well.

[Though the duration of symptoms is not given, nor the presence or absence of teeth remarked with the statement concerning the lack of changes in the gums, there is vague reference to the existence of ecchymoses late in the course of the disease, and it may be assumed from the context that the condition lasted some days, at least, before antiscorbutic treatment was begun. The rapid improvement is most suggestive of the correctness of the diagnosis. Infantile scurvy is so generally understood by medical men at the present

day, that incipient cases are frequently met, recognized, and cured before gross changes have taken place. In several cases in which we have had reason to be on the watch for scorbutic symptoms we have seen sponginess of the gums appear and yield to treatment all within a week, without any of the grosser symptoms having time to manifest themselves.]

The Duration of Immunity Conferred by a Single Injection of Diphtheria Antitoxin.—F. GORDON MORRILL (*Boston Medical and Surgical Journal*, March 3, 1898, p. 193), whose experience in immunizing a large number of children in the Boston Children's Hospital is already well known, makes the following statements in concluding a paper on this subject: Immunity in any given case, of no matter how thorough exposure to diphtheria, may be conferred for at least ten days by the injection of a small dose (100 to 250 units) of serum, provided it is given twenty-four hours previous to actual infection. A larger dose (250 units for a child of two years, up to 500 units for one of eight or over) will confer safety for three weeks—or, to be a little more conservative, let us say, twenty days—under similar conditions.

No harm will result from the treatment in a vast majority of cases of *sick* children, and probably in no case of a healthy child, provided the serum used is up to the present standard of purity.

The Treatment of Epidemic Cerebro-spinal Meningitis with Corrosive Sublimate.—DINAMI (*La Pediatria*, November, 1897) has employed with success the method of Dazio in treating meningitis with hypodermatic and intravenous injections of mercuric chloride. The patient was a girl of seven years, seized suddenly with high fever and intense cephalalgia. For five days the temperature ranged between 102° in the morning and 104° to 105° in the evening. There were general prostration, arrhythmia of pulse, scaphoid belly, vomiting, deafness, and photophobia. After the sixth day the fever became intermittent in character, ranging from 97° in the morning to 104° or 105° at night, the general state being quite good during the apyrexia, but with the rise of temperature coma being present. On the seventeenth day, after failure of treatment with calomel, wet cups, cold applications to the head and neck, opiates, antipyrin, phenacetin, and other remedies, one centigramme of sublimate was injected beneath the skin. After the first injection the temperature fell half a degree, and the headache became less intense. After the fifth injection fever and headache disappeared completely. Convalescence was slow, but the child was able to leave her bed fifteen days after the cessation of fever. The power of walking was not regained for a month, and during convalescence hallucinations were present. Despite the use of iodides the deafness persisted.

Noma in the Course of Pertussis.—An instance of noma complicating pertussis, one of the rarest of the associations of cancrum oris, is recorded by SIMONINI (*La Pediatria*, November, 1897). The patient was a girl of three and one-half years. Toward the end of a severe attack of pertussis lasting three months, the parents noticed dribbling of saliva and detected a swelling of the gum over the right canine and premolar teeth. Two days later the disease had spread to the lip, the mucous membrane became livid and covered

with grayish vesicles, which ulcerated; the teeth became loose and the breath fetid. The child was first seen on the twelfth day of the disease. The right labial commissure was totally destroyed for an extent of two centimetres. Within the mouth the gangrenous lesion was more extensive; two teeth had been lost, the canine and premolar first involved; the mucous membrane was spongy, sanguinolent, and black; the frænum of the tongue was ulcerated. The temperature was 100.4°. Cure followed the use of the actual cautery, aided by local antiseptic applications.

HYGIENE AND PUBLIC HEALTH.

UNDER THE CHARGE OF

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Aerated Butter.—The unequal quality of Irish butter, due to the very different degrees of skill and care, we might add of cleanliness, in its preparation, is well known in the markets, good and bad strata not infrequently being met with alternating in the same cask. To overcome this a company has been formed in Dublin to work a process substantially the same as that of R. Andrews' patent (No. 4622 of 1894), much used for the purification of other fats. Rancid butter is emulsified with buttermilk at a regulated temperature, and is then subjected to the continued action of a much divided current of warm air, which carries off the volatile acids to which the rank taste and odor of bad butter are due, non-volatile impurities being at the same time washed out of the fat. After the two or three hours required for this process, according to the quality operated on, the temperature is lowered, and the purified butter is repeatedly washed with cold water, the product being perfectly sweet and well granulated, with an agreeable flavor; in fact a really high-class article. Dr. Emerson Reynolds, F.R.S., Professor of Chemistry in Trinity College, Dublin, testifies to the unqualified success of the procedure in transforming bad butter into a wholesome and perfectly unobjectionable food.

Tuberculosis in Fish.—MM. DUBAR, BATAILLON and TERRE communicated to the Academy of Medicine an observation, as yet unique, of the occurrence of tuberculosis in a cold-blooded animal. The circumstances were peculiar, constituting, in fact, an undesigned experiment. The dejecta and sputa of a woman suffering from advanced pulmonary and intestinal tubercle, having been for some time regularly emptied into a pond in which were large numbers of fine carp, many of the fish were found swollen and dead, and on examination their livers, spleens, and other viscera were seen to be stuffed

with tubercle bacilli. Healthy fish fed with this tubercular material, or with that from tuberculous guinea-pigs and fowls, succumbed in like manner within three or four weeks, but the fish tubercle proved quite innocuous to warm-blooded animals. The obvious conclusions to be drawn from these facts are that fish are even more susceptible to tubercle than rodents, etc., and owe their exemption to the conditions under which they usually live; that if man be as resistant to fish tubercle as other warm-blooded animals have shown themselves to be, there is little danger from the consumption of diseased fishes, which, moreover, are not likely to be met with under ordinary circumstances, since the effect of the cultivation of the bacilli at the low temperature of the fish's body has been the attenuation of their virulence, but whether they would regain it in time in the warm blood of a higher animal has yet to be seen; very probably they would.

Tetanus in "Made Soil."—Ever since 1884, when Nicolaier, following up the discovery of the bacillus of tetanus by Carle and Rattone in the dust of old buildings, found it in the soil of gardens, stable-yards, etc., it has been known that the natural habitat of the bacillus is in the outer world, whence it gains access to the body by the inoculation of "dirt" in which it is already present; and that the frequency with which tetanus follows wounds of the sole of the foot and the palm of the hand is owing simply to the fact that such wounds are specially exposed to the entrance of earth and dirt from the conditions under which they occur. But, as might have been expected, "made soil," as it is called, the mixture of house refuse, street sweepings and mud, with ashes and other materials, organic and inorganic, has been found by Dr. K. Kratz to be above all others rich in these bacilli, and therefore specially dangerous. In the course of an exhaustive investigation into the chemistry and bacteriology of the soil composing the "tips" of the municipal dust-yards at Giessen, he found that the injection of the watery extract of such soil into mice was invariably followed by death from tetanus, though negative results only were obtained in control experiments with the earth from an adjacent grass meadow. The importance of this observation is obvious when we consider the extent to which such "made soil" is used in building operations for filling up natural depressions or those produced by the excavation and removal for sale of good sand and gravel, and that the sites of dust-yards, when, through the growth of towns, they become surrounded by houses, usually pass into the hands of the builder, the authorities seeking others more remote.

Deaf Mutism.—Holding that all philanthropic effort having as its aim the amelioration of the material and social conditions of the community comes within the scope of public health, it is with mingled feelings that we read the report of the recent "Congresso pei Sordomuti" at Milan. If in Italy the deaf mute have been relegated by the statute law to the category of idiots and imbeciles, incapable of exercising civil or political functions, his position in Great Britain has been a peculiarly anomalous one, since without any disability or exemption he has either been left uneducated, or has received an instruction fitting him for intercourse with such only as were afflicted in like manner, narrowing his capacities and tending to the perpetu-

ation of deaf mutes through intermarriage. In Germany the method of finger language is quite obsolete, having everywhere given place to that of teaching the deaf mute to emit vocal and articulate utterances, which he does not hear, and to interpret by sight the lip-movements of those possessed of speech, a system which, notwithstanding its great and obvious advantages, has made but very slow headway here. The most interesting feature in the Congress was the object-lesson presented of the capabilities of the deaf mute for taking his part in social and public life, if only the exercise of his power and the growth of his intellect be not stifled by neglect. Infants and children chattered according to their abilities, one or two deaf mutes took part orally in the discussion, a vote of thanks was moved in a graceful speech by another, and the pupils of one of the forty or fifty institutions in the kingdom, 100 strong, after going through some well-executed evolutions sounded round after round of loud "evvivas!" It is remarkable that the only country in which these unfortunate persons are deprived by the law of their civil rights should be that in which most has been done to fit them to take their place among educated citizens.

Cholera-infected Food.—According to the *Pharmaceutical Journal* of July 31 and August 7, 1897, a number of rabbits that had been inoculated with cholera and other pathogenic bacteria were taken from the Aubervallier's Hospital in Paris by thieves, who carted them about the suburbs of Clichy and St. Ouen, and sold them at low prices. The police succeeded in finding several of the people who had purchased, but only one family that had eaten them. In this case no trouble of any sort was experienced.

Disinfection by Alcohol.—According to DR. F. EPSTEIN (*Zeitschrift f. Hygiene und Infectionskrankheiten*, xxiv. p. 1), alcohol is strongest in disinfecting power at 50 per cent., from which point in either direction its power diminishes. Absolute alcohol has no power at all. Chemicals which are more or less efficient in aqueous solution lose their disinfectant property in strong alcohol; but corrosive, carbolic acid, lysol, and thymol have greater effect in 50 per cent. alcohol than in water.

Widal's Test for Typhoid.—The expectations raised some few years ago, when Widal announced his discovery that the serum of a patient suffering from typhoid fever, added to a broth culture of the bacillus of that disease, caused the bacilli to agglutinate in groups or masses, to lose their mobility, and to die, seemed doomed to disappointment by the subsequent observation that the same phenomenon might be produced by the serum of other febrile diseases. It was true that the positive results, while apparently constant in enteric fever, were exceptional in non-typhoid cases, but the exceptions were numerous enough to cast a doubt on each single observation and to deprive the test of all certainty and diagnostic value. It was next found that in cases of true enteric fever the delicacy of the test was enhanced by the use of a larger proportion of the culture fluid, or, still better, by the dilution of the serum with water or broth. Widal himself adopted dilutions of 1 : 10, with better results in cases of true enteric fever, but without eliminating erroneous conclusions in others. Subsequent observers, however, ascertained that

by carrying the dilution further a point was reached at which the serum of other diseases ceased to react, though the activity of that of enteric fever was unimpaired.

Mesnil de Rochmont, from a large number of observations, came to the conclusion, fully confirmed by others, that the highest dilution in which a positive reaction could be obtained in any non-typhoid case was 1 : 25; and consequently 1 : 30 was given by Gruber and Stern, 1 : 40 by Förster and Kühnau, and 1 : 50 by Sklower and Scholtz, as results which would effectually exclude the possibility of error. Scholtz, having verified the observations of previous observers, especially Fraenkel, Stern, and Förster, and investigated exhaustively several hundred cases of enteric fever and others more or less closely simulating it, lays down the following propositions:

1. That in cases of true enteric fever dilutions of 1 : 50 will generally afford positive indications within the first, and always during the second week, the energy of the serum increasing as the progress of the disease.

2. That the serum retains this property for many months or years; in fact so long as immunity, with which it is in some way connected, persists.

3. That apparently positive results are afforded in a certain proportion of febrile diseases other than enteric, but that this occurs only when the serum is comparatively concentrated, and never in dilutions of 1 : 30 or more.

4. That if, in the case of a patient suffering from a febrile disease of a doubtful character, a positive result be really due to the fact of his having passed through an attack of typhoid some years previously, a second examination made after the lapse of a week will not show the increase in the activity of the serum which would have been observed had the actual illness been genuine enteric, of however mild a type. The degree of dilution which such cases will bear depends chiefly on the time that has elapsed since the attack; in those of very recent date it may be as high as in cases of present enteric fever, but it will remain stationary through the whole course of the actual illness.

Thus, among one hundred cases of non-typhoid febrile disease, Scholtz obtained apparently positive results in nineteen with dilutions of 1 : 5; in eleven of these with 1 : 10; in seven with 1 : 15; in three with 1 : 20; and in one a very faint reaction with 1 : 25; whereas, in as many of true typhoid, he never failed with dilutions of 1 : 50.

Examining the blood of six persons who had suffered from enteric fever at periods ranging from eight to fifteen years previously, he obtained positive results in all with dilutions of from 1 : 25 to 1 : 12; and in some of more recent date with 1 : 40, though in perfect health at the time.

The highest dilution with which he observed a fallacious positive result in non-typhoid febrile attacks was, as stated above, 1 : 25, or a distinct reaction with 1 : 20; whereas, on the other hand, the lowest dilution with which the reaction was lost early in the course of a mild attack of real typhoid was 1 : 45, and in this a later observation was successful with 1 : 50, the difference between 1 : 25 and 1 : 45 giving a margin wide enough to exclude all risk of error.

The test is equally available after death, and in one case with no characteristic symptoms during life, a post-mortem application gave a positive result with 1 : 50, which was confirmed by the seurm revealed at the autopsy.

The procedure he recommends is to keep the broth culture of the bacilli for the six hours preceding the examination at a temperature of 37° C., so as to secure their active growth; to use as a diluent a simple sterilized broth, and, rejecting all elaborate apparatus, to employ only a capillary pipette graduated to measure $\frac{1}{100}$ c.cms.

He begins by making a 1 : 10 dilution of the serum and adding it to an equal volume of the culture. If the result be negative, nothing more is needed; but if positive, he tries the effect of 1 : 40 and 1 : 50, the indications of which, whether positive or negative, are to be taken as conclusive, unless there be grounds for suspecting that the reaction may be owing to a remote attack, or that the disease be not further advanced than the first week; in either of which cases the examination should be repeated a week or so later.

Microbe of Acute Rheumatism.—The frequent association of acute rheumatism or rheumatic fever (neither name being satisfactory in the light of modern views as to the etiology and relation of the so-called acute and chronic rheumatism) with cerebro-spinal meningitis, now acknowledged by all to be a bacterial and infectious disease, and with tonsillitis, many forms of which are certainly so, has for some time suggested the possibility of its being, like malaria, parasitic, though not communicable.

Pierre Achaime, so long back as 1891, in a communication to the Société de Biologie, described an organism which he had found in the blood of a man suffering from rheumatic fever with cerebral complications, and which he believed to be pathogenic, giving it the name of *Thiroloix Achaimii*. In the *Annales Pasteur* of November, 1897, he states that he has since been able to identify it in eight cases, and that it has been observed also by Lucatello at Genoa and Riva at Parma in this disease, but in no other. In two fatal cases he found it in enormous numbers in the cerebro-spinal fluid, where it was present as an almost pure culture; but that its presence in the blood of the living subjects could be demonstrated only by artificial cultivation in a mixture of bouillon and milk maintained for eight to ten days at temperature of 37° C. under anaërobic conditions. Its form varied considerably with the culture media employed, but it grew better in liquids than in solids and at a temperature of 35° to 38° C., the extreme limits being 25° and 40° C.

Growth of Bacteria in Sterilized Milk.—Milk when drawn from the udder of a healthy cow is perfectly sterile, and all the so-called "spontaneous" changes of souring, curdling, and putrefaction are the results of the introduction of successive contingents of bacteria, beginning with B. coli, in the process of milking, which multiply until in the shops they number tens of thousands or even millions in the c.cm. Sterilizing the fresh-drawn milk, besides destroying any pathogenic germs that may chance to be present, delays the souring until after the vessels have been opened, but recent observations of SCHOTTELIUS show that it does more, and that the proliferation of bacteria proceeds with but a tenth part of the rapidity exhibited in fresh unboiled milk under identical conditions; at any rate, this was the case with those of diphtheria. He added 1 c. cm. of a bouillon culture to 2 c. cms. of (1) fresh milk drawn from the cow with every precaution, and therefore sterile, (2) sterilized milk, and (3), by way of comparison, sterilized bouillon; one

set of tubes having been kept at the ordinary temperature of the room, and the others at that of the body; the enumerations were made at the end of six hours. The exact number of bacteria introduced is not stated, but uniformity was secured by the inoculations being performed simultaneously from a culture that had been well agitated.

The results were: Raw milk, 21,280,000, at 15 per cent.; 50,160,000, at 37 per cent. Bouillon, 7,600,000, at 15 per cent.; 18,240,000 at 37 per cent. Sterilized milk, 2,200,000, at 15 per cent.; 6,080,000, at 37 per cent.

Alcohol in Milk.—The effect of brewers' grains, distillery swill, ensilage—in short, of fermented fodder of any kind—on the milk of cows in rendering it prone to turn sour within twelve hours and to set up gastro-intestinal disturbance in infants is so well known that the use of such foods is expressly prohibited in the contracts between the great dairies, the condensed-milk companies, etc., and the farmers from whom they obtain their supplies; but whether alcohol ever passes over as such into the mammary secretion of the cow or of the human female is a point that has been denied on negative and maintained on insufficient evidence. A recent observation by H. WELLER has, however, proved the possibility of such a result beyond question, for he was able to recover 0.96 per cent. of alcohol from the milk sent out from a dairy owned by the proprietors of a large distillery, where, in addition to cake and other fodder, the swill, containing 5.9 per cent. of alcohol, or as much as is present in strong ales, was given to the animals, though in what quantity is not stated. The milk examined was perfectly fresh and otherwise of normal composition, without a trace of souring. The specific gravity was 1.0335, the total solids 13.307, and the fat 3.79 per cent. It had, however, a peculiar harsh taste, of which the consumers complained, and which Weller found to be due to a flocculent substance present in the distillery waste and in the milk, the nature of which he does not seem to have taken any means to determine.

Transmission of Infectious Diseases through the Air.—DR. EDUARDO GERMANO, continuing his researches on this subject, the first of which were referred to in the March number of this JOURNAL, finds that the diphtheria bacillus (*Zeitschrift f. Hygiene und Infectionskrankheiten*, xxv. p. 439) withstands long drying in membranes, tissues, and dust, even when the drying process is assisted by sulphuric acid, and that its resistance is greater according to the amount of the enveloping material which retards oxidation. When completely dry it preserves its full virulence up to the time it dies. He concludes that the air can disseminate the bacilli in an active condition in the form of dust. As to erysipelas, pneumonia, and other streptococcus infections, he finds (*Ibid.*, xxvi. p. 66) that the resistance of streptococcus to the drying process, while varying according to the method followed and to the enveloping material, is always high, and may last a number of months, and that the air may be credited with an important share in the transmission of infections. The diplococcus in general can bear drying for a long time, but some varieties show but little resistance, and, as a rule, it lasts better dry than moist. Its duration of life is not affected by the rapidity of the drying process with medium temperature. While it has not the resistance shown by the streptococcus, its dissemination through air must be regarded as possible.

His concluding paper (*Ibid.*, xxvi. p. 273) deals with cholera, plague, and epidemic cerebro-spinal meningitis. Contrary to the views of Pettenkofer and his school, who hold that air is the only medium for disseminating cholera, and that water and moisture have no important part in the process, his experiments lead to the conclusion that the cholera organism retains its virulence only so long as it remains moist, and dies quickly on drying, and the more so if the process is hastened. In his experiments he employed dust impregnated with bouillon cultures, and sterilized feces, which he infected with the organism and then mixed partly with fine sand and partly with brick-dust. He places the cholera organism in the class of bacteria which show least resistance to drying, and concludes that dissemination by air is most highly improbable. His work on the bacillus of plague agrees in results with that of Kitasato and of Wilm. It does not withstand drying, but lives a long time in the moist condition. It remains active fairly long when dried on cloth, perhaps because then complete drying requires a long time, and thus may be explained the danger of infection which is recognized to exist in infected clothing. His experiments with the diplococcus of epidemic cerebro-spinal meningitis confirm the conclusions of Jäger as to its resistance to drying. Jäger found the organism in an active condition in a handkerchief six weeks after use by a patient sick with the disease. Germano shows that it belongs to the class of bacteria which show the greatest possible resistance to drying, whether the process is quick or slow or assisted by the action of sulphuric acid, and concludes that it may without difficulty get into the air in the form of dust, and thus spread the infection.

Dr. Max Neisser (*Ibid.*, xxvii. p. 175), working in the same line with an apparatus of his own design, which establishes a constant aspiration current of dusty infected air, disagrees with Germano in his conclusions that the pneumococcus withstands drying, inasmuch as while mice inoculated as controls with infected dust died from the infection without exception, negative results were obtained in every one of twenty-four inoculations with dust that had been disseminated through the apparatus in the current of air. His experiments with various organisms led him to conclude that dust-infection is impossible with the organisms of diphtheria, typhoid fever, plague, cholera, and pneumonia, but possible with staphylococcus pyogenes aureus, B. pyocyaneus, B. anthracis, B. tuberculosis, and meningococcus.

The Typhoid Bacillus and Buttermilk.—Drs. Eugene Fraenkel and J. Kister (*Münchener medicinische Wochenschrift*, February 18, 1898), having reason to believe that the unusual amount of typhoid fever at Hamburg during the summer of 1897 was due in part to infected buttermilk, undertook the study of the question whether the *B. typhosus* can exist in that fluid, concerning which point there is more or less of conflicting testimony. Obtaining some samples, they first investigated the number and identity of the contained bacteria, and learned that while the number varied widely, the species were always about the same. Finding no pathogenic organisms, they sterilized specimens in test-tubes a half hour a day for three days, then planted the typhoid bacillus in them and kept them at different temperatures: on ice, at 22° C. and 37° C. Loops were taken from each from time to time and planted, and all yielded positive results. The specimen kept at room-

temperature was under observation nine days; the others were not examined after the third. Then, specimens of fresh buttermilk containing all its bacteria were planted and kept under the same conditions, and from them the same results were obtained. Yet there was this difference, that there was always a diminution in the number of the pathogenic organisms, and this was more marked, and sometimes very rapid, with increasing temperatures.

How the Color of Drinking-water Due to Humin Substances may be Influenced by Iron Compounds.—W. SPRING (*Chemisches Centralblatt*, 1898, i. 410) advances as a result of some observations on the color of drinking-waters an explanation of the influence of iron salts. Mixing brown peaty water with dilute solution of ferric salts, it will be noticed that the color is at first dark, but under the influence of light becomes less and less so, and meanwhile a precipitate settles out. The humin substances are oxidized and unite to form insoluble compounds with metallic oxides. In this process the ferric are reduced to ferrous compounds having insignificant color, but the latter are soon again oxidized to ferric, and then resume the separation of the organic coloring. He likens the function of iron in the purification of water to that of the hæmoglobin of the blood.

PATHOLOGY AND BACTERIOLOGY.

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Cancer of Œsophagus with Involvement of Heart and Lung.—BUREAU (*Bull. Soc. Anat. de Paris*) reports the above condition in a man, fifty years of age, emaciated and cachectic, with pain on swallowing, and vomiting, which only once showed the presence of blood. Later a pleurisy developed and 500 c.c. of fluid were withdrawn. Sputum became fetid. Tachycardia was observed, the pulse on one occasion being 160. Post-mortem examination showed a constricting cancerous growth of the œsophagus with ulceration and perforation into the middle lobe of the right lung. The œsophagus was adherent to the left auricle of the heart with projection of the growth into this cavity, and there was a formation of cancerous nodules the size of a pea on the endocardium. The muscular fibres of the heart were atrophied and also infiltrated with cancer cells. The left inferior pulmonary vein was compressed by the growth, as was also the right pneumogastric nerve. Histological examination of the growth showed it to be an epidermoid cancer.

The Pathology of Tabes Dorsalis.—In an exhaustive monograph on the pathology of the spinal cord changes of locomotor ataxia, REDLICH presents

a critical review of the literature of this subject, and adds much of interest derived from personal studies of his own, made for the most part in the laboratory of Professor Obersteiner (*Die Pathologie der tabischen Hinterstrangserkrankung ein Beitrag zur Anatomie und Pathologie der Rückenmarkshinterstränge:* Jena, 1897: G. Fischer. Pp. vi. 205).

The work is divided into four parts, devoted respectively to the normal anatomy and histology of the posterior columns, to their pathological changes in locomotor ataxia, to the etiology of locomotor ataxia as gathered from statistics and from clinical studies, and to the pathogenesis of the lesion in the posterior columns incident to the disease.

His studies of the distribution of the structural changes lead Redlich to renounce the theory, advanced by Strümpell, that the lesion of tabes is a system degeneration. The opinion of Flechsig, that it follows the course of the developmental tracts, is also denied, and it is maintained that the first evidences of degeneration are to be found in the posterior roots, and that the degeneration is rather segmental than systemic in its occurrence and distribution.

The part of the posterior roots which seems to be earliest affected is the region of their union with the spinal cord. It is maintained that this region is more liable to injury because of certain peculiarities of its position, and in support of this view the acute angle at which many of the lower sensory roots unite with the cord, and the opportunity thereby afforded for constriction of the roots by the pia, are brought out. Actual meningitis is believed to be the cause of the local degeneration in comparatively few cases, as are also pressure from inflammatory lesions in the neighboring bones or from changes in the bloodvessels at the point of entrance of the roots into the cord. Nor do there appear to be primary changes in the ganglia of the posterior roots, nor in the peripheral nerves. Thus, while Redlich is convinced that the lesion of tabes has its origin in the posterior roots just at their point of union with the cord, and that this is anatomically a *locus minoris resistentiæ*, he is unable to decide what is the precise exciting cause of the change.

As regards a more remote etiological factor syphilis is noted as having been present in 65.2 per cent. of the men (seventy-two), and in 23.4 per cent. of the women (thirty) studied by Redlich. In a few cases other infectious toxic conditions seemed to have taken the place of syphilis as an etiological factor.

The monograph is handsomely illustrated by a number of well-executed plates, and will repay a careful perusal.

Experimental Studies in Cardiac Arrhythmia.—M. HEITLER (*Wiener klin. Wochenschrift*, 1898, Nos. 3 and 8). Arrhythmia is a recognized symptom of cardiac disease, but its exact significance has only recently been investigated. The author has made experiments upon curarized dogs, the pulse being recorded in the usual manner upon the kymograph. It was first found that any mechanical irritation of the heart would produce arrhythmia, and that special cardiac regions were more susceptible; for example, the longitudinal sulcus, especially its upper third. Arrhythmia was more intense if the irritation was applied during systole than diastole. Cutting the vagi had no effect upon the arrhythmia following irritation. The lightest touch of the visceral pericardium caused as much arrhythmia as deep stimulation. Cocaine applied to the pericardium overcame the irritation and no arrhythmia resulted. Seizing

the heart with pincers caused arrhythmia at the first touch and upon removal, not for the entire period. Irritation of the endocardium produced arrhythmia in the same way as the pericardial irritation. This was proved by passing a tube with a sliding rod inside into the left auricle. The endocardium was irritated by moving the rod back and forth, without producing any lesion of the cardial walls or at most only a slight erosion. Irritation in the neighborhood of the valve produced greater arrhythmia than irritation of the deeper parts. Both sides of the heart react alike. Myocardial irritation was studied by thrusting electrodes varnished to the tips into the heart-wall. No arrhythmia resulted from the mechanical irritation of the electrodes, and it was only upon using a very powerful electric stimulation that arrhythmia resulted, and here the contractions as registered by the sphygmograph differed markedly from those of pericardial and endocardial irritation. Mechanical irritation of the myocardium failed completely in producing arrhythmia. A piece of the heart-muscle could be snipped out without producing arrhythmia.

Primary Cancer of the Appendix.—Mosse and Daunic report, in the *Bulletin de la Soc. Anat.*, November, 1897, a case occurring in a woman fifty years of age, who died of cardiac disease. The appendix floated freely in the peritoneal cavity; was 4 cm. in length, and 4.5 cm. in circumference, and cylindrical in shape. On transverse section the muscular layers could be seen enclosing a hard mass, within which was a narrow canal opening freely into the cæcum. The tumor did not project at all into the cæcum, but was limited to the appendix. Histological examination showed the muscular layers for the most part normal, in places separated by infiltrated epithelial cells forming small islands. The peritoneal coat was normal. In the central part of the tumor, near the central canal, were seen remains of the mucosa and of Lieberkühn's glands. The latter had for the most part undergone cancerous transformation and were probably the origin of the neoplasm. In places the neoplasm presented the appearance of cylindrical-cell cancer. This condition of the appendix, with absence of cause in the cæcum, lymph-nodes, and other viscera, warranted the assumption that the growth was primary cancer of the appendix. The observers note the exceeding rarity of the primary tumors of this diverticulum of the intestine. Of seventeen cases of primary tumor collected by Lafforgue in the study of the subject, nine were cancers, one lipoma, and one myoma.

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CONTENTS.

ORIGINAL COMMUNICATIONS.

PAGE

Antitoxin Treatment of Pneumonia. By ANDREW H. SMITH, M.D. . . . 377
Primary Malignant Disease of the Suprarenal Bodies. By H. D. ROLLESTON, M.A., M.D. Cantab., F.R.C.P., and H. W. J. MARKS, M.A. Cantab., M R.C.S. . . 383
Anomalous Position of the Colon; with Report of a Case Discovered by Exploratory Operation. By JOHN B. SHOBER, A M., M.D. 405
Two Attacks of Temporary Hemiplegia Occurring in the Same Individual as the Result of the Use of Peroxide of Hydrogen in a Sacculated Empyema (Pleural). By E. G. JANEWAY, M.D. 420
Gastric Syphilis, with Report of a Case of Perforating Syphilitic Ulcer of the Stomach. By SIMON FLEXNER, M.D. 424
The Diagnosis of Nephritis without Albuminuria. By ARTHUR R. EDWARDS, A.M., M.D. 432
The So-called Hyaline Bodies and other Cellular Degenerations in Nasal Polypi. By JONATHAN WRIGHT, M.D. 445

REVIEWS.

Treatise on Clinical and Operative Surgery. By Le Dentu and Delbert . . 456
Atlas and Abstract of the Diseases of the Larynx. By L. Grünwald . . . 457
Disorders of the Circulation and their Treatment by Baths and Gymnastics. By Dr. S. Ch. Graupner 458
Handbook of Human Anatomy. The Ear. By Karl von Bardeleben . . . 458
The Cæcal Folds and Fossæ and the Topographical Anatomy of the Vermiform Appendix. By Richard J. B. Berry, M.D., F.R.C.S , F.R.S. Edin. . . . 459
Atlas of Methods of Clinical Investigation. By Dr. Christfried Jakob . . . 470
Practical Guide to Urinalysis, with an Appendix on the Analysis of Stomach-contents. By Prof. Lassar-Cohn 460
An American Text-book of Genito-urinary Diseases, Syphilis, and Diseases of the Skin. Edited by L. Bolton Bangs, M.D., and W. A. Hardaway, A.M., M.D. . 461
The Psychology of Suggestion: A Research into the Subconscious Nature of Man and Society. By Boris Sidis, M.A., Ph.D. 463

PROGRESS OF MEDICAL SCIENCE.

THERAPEUTICS.

PAGE

Pyramidon in Nervous Diseases . 465
Oil of Salosal in Urinary Diseases . 465
Rhus Aromatica in Incontinence of Urine 466
Value of Certain Drugs in Gout . . 466
The Melaleuca Viridiflora . . . 467
Clinical Observations upon Kryofine 468
Peronin 468
Eucaine-B in General Surgery . . 468
A Clinical Study of Kryofine . . 468
Dry Calcium Sulphohydrate . . 469
Treatment of Latent Dyspepsia . . 469
Infiltration Anæsthesia . . . 469
Eucaine-B 470
Treatment of Gonorrhœa . . . 470
Yellow Palms a Sign of Typhoid Fever 470
Exercise and Carbonated Brine Baths in Chronic Heart Diseases . . 470

MEDICINE.

	PAGE		PAGE
Incubation Stage of Typhoid Fever	471	Left Ventricle in Mitral Disease	474
The Diazo-reaction	472	Papillary Myxoma in Tricuspid Valve	474
A Crural Venous Murmur	472	Dulness Over the Apices of the Lungs without Pathological Changes	474
Two Rare Cases of Auto-intoxication	473	Pericarditic Pseudo-cirrhosis of Liver	474
Bacterial Complications of Diabetes	473		

SURGERY.

Hæmatemesis Consecutive to Exulceration of the Stomach	475	Tibio-tarsal Disarticulation	479
Typical Form of Intestinal Obstruction	476	Surgical Treatment of Filarial Disease	480
Operation of Gastro-enterostomy	477	Amputation of Head of Femur	480
Method for Controlling Hemorrhage	477	Treatment of Chronic Empyema	481
Abdominal Incision	478	Lymphatic Disease of Testicle	481
Treatment of Flat-foot	479	Employment of the Murphy-button	482
		Treatment of the Movable Liver	482

OTOLOGY.

Congenital Malformation of Auricle	483	Acute Otitis Media in Children	484
Faucial, Nasal, and Aural Diphtheria	483	Indications for Trepanation of the Mastoid Process	485
Pneumo-massage of Ear	483		
Total Excision of Membrana Tympani	484	Otitic Cerebral Abscesses	486

DERMATOLOGY.

Erythema Induratum of Bazin	487	The Radical Treatment of Lupus	489
Lichen Scrofulosorum	488	On Miliary Tuberculosis of the Skin	489
Herpes Zoster	488	Pyrogallol in Tuberculous Processes	490
Some Undescribed Forms of Herpes Tonsurans	488	Monochlor-acetic Acid in Xanthoma	490
Ringworm as it Appears in Boston	488	Urticaria with Recurrent Hæmatemesis	490
Dermatoses Albuminuricæ	489	Lactophosphate of Lime in Acne	490
		New Human Parasite of Sarcoptic Tribe	491

OBSTETRICS.

Intra-uterine Use of Colpeurynter	491	Intra-uterine Typhoid	494
The Etiology of Hydramnios	492	Osteomalacia and its Treatment	494
Appendicitis Complicating Pregnancy	492	Rupture of the Uterus—Recovery	495
Ophthalmia in the Newly-born	492	Superfœtation in the Human Race	495
Partial Occlusion of Pelvic Brim	492	Contracted Pelvis	495
Labor Complicated by Myoma of Womb	493	Influencing the Sex of the Fœtus	496
Neglected Shoulder Presentation	493	Gunshot-wound of Uterus and Child	496
Seventy-four Years' Experience in the Induction of Labor	493	Incision of Fundus in Cæsarean Section	496
		The Pathology of Osteomalacia	496

GYNECOLOGY.

Treatment of Fibromyoma	497	Vaginal Hysterectomy for Fibromyoma	498
Primary Carcinoma of Fallopian Tube	497	Results of Vaginal Hysterectomy for Diseased Adnexa	499
Cyst of the Round Ligament	497		
Gall-bladder Adhesions	497	Results of Curettement	499
Oöphorectomy in Inoperable Cancer of the Breast	498	Metrorrhagia in Children	499
		Pelvic Abscess of Appendicular Origin	499

HYGIENE AND PUBLIC HEALTH.

Fluoride of Sodium as a Food Preservative	500	New Constituents of the Atmosphere	501
Typhoid Fever and Infected Ice	501	Industrial Hygiene—Matchworkers	501
		Disinfection by Formaldehyde	502